Mechanical Ventilation
MADE EASY ®

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Forewords

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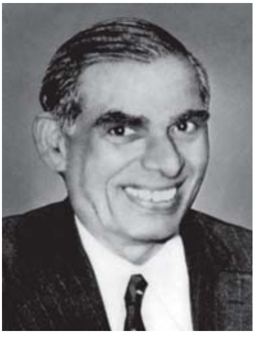
To

Prof (Dr) AA Asirvatham

- My Godfather who loved me and guided me in my personal as well as professional life
- A wonderful human being with a very kind and loving heart towards his patients and his fellow men
- A loving teacher with great virtues and skills, held in high esteem in the field of surgery, responsible for transforming thousands of medical men into good surgeons



Prof (Dr) AA Asirvatham
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1961–1973
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Foreword

There has been a need for a simple textbook on Mechanical Ventilation which can be read and understood easily not only by clinicians but also by paramedical staff working in Intensive Care Units.

As clearly averred by the author Prof S Ahanatha Pillai, the available books were of a high standard which can be understood by the Intensivists, the Anesthesiologists, or the Pulmonologists. All these specialists may not be available at the spot of acute crisis. The patient's condition would deteriorate further by the time these specialists arrive. This book would help the clinicians on the spot as well as the paramedics in tackling the emergency situation.

No ventilatory therapy is complete unless one knows how to wean the patient from the ventilator. This has been meticulously dealt with by the author. Further, the book is written in a simple language and at the same time avoiding sophisticated technical terminologies.

True to commitment in imparting knowledge to students and clinicians, Prof S Ahanatha Pillai has taken great pains to write this important topic in a simple way.

I strongly recommend this book to be kept in the libraries of all the medical institutions in general, as well as in the libraries of Intensive Care Units and Ana esthesiology Departments.

Prof (Dr) E Radhakrishnan MD DA
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FOREWORD

It is a great pleasure to go through the book *Mechanical Ventilation Made Easy* by Dr S Ahanatha Pillai written especially for the clinicians and the people working in Intensive Care Units.

It makes an enjoyable reading of a relatively difficult subject. The text is presented very clearly starting from the applied anatomy and physiology, gradually building up to mechanics of breathing and then to the clinical applications of mechanical ventilation. All the essential aspects are discussed in a very simple way that makes it easy to understand.

Though the author has mentioned that he has avoided intricate technical details, I find that it contains almost all essential details.

I hope this book will be useful to all those who work in Intensive Care Units and who wish to gain in-depth knowledge about mechanical ventilation.

I am sure this small book on Mechanical Ventilation will certainly serve the purpose for which it is written.

Prof (Dr) J Renganathan MD DA
Former Professor and Head
Department of Anaesthesiology and
Intensive Respiratory Care Unit
Government Stanley Medical College, Chennai
National Secretary
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Preface

Numerous books are available in the market on Mechanical Ventilation and Ventilator Therapy. These books are too good for any specialist to read, but are of high standard for the students. Many internees and nurses just look at a ventilator, as if it is a monster and show the least interest to know anything about it. On the other hand, there are many young doctors and nurses having a lot of curiosity to learn about ventilators. They search for a simple book that tells them clearly all about the mechanical ventilation. This book is meant to fulfil their requirements.

Nowadays, non-availability of ventilators is no longer a problem. The most sophisticated ventilators are available everywhere and are in wide use across the world. The patients on ventilator therapy are managed by specially trained and efficient nurses who are well versed in ICU nursing, under the guidance of Intensive Therapy Physicians.

Nevertheless, many a time, the initial management of a patient which includes intubating the patient; connecting him to the ventilator and making the initial ventilator settings are all done by the intern, the house surgeon or the nurse in the Intensive Therapy Unit. Therefore, it is essential that these young doctors and nurses have a clear orientation of mechanical ventilators for a better management of their patients.

However, unfortunately, some of the doctors and nurses lack the information about the fundamental principles involved in ventilatory support. They have very little idea as to how different modes and settings are meant to support the ventilation. The application of suitable modes and settings for a particular patient in a particular clinical situation requires a better knowledge of these fundamentals. It is needless to say that if there are any errors at this primary level, the whole purpose of the therapy may fail.

This small book is designed in such a way that it is directed towards filling this lacuna of information.

It gives details of two aspects:

- The basic mechanisms of normal respiration and how they are modified in artificial ventilation
- How the different modes and settings of ventilator work with their indications, merits and demerits

Purposely, I have tried to present the material in a simplest style possible and in a very simple language. I have provided the essence of the subject without going deep into the intricate details so as to make it easy to understand. Too much of technical details are deliberately avoided, as it may confuse the students and stop them from reading further. Only to make the reading interesting, I have given the history as the second chapter.

I hope this book will be of definite use to medical internees, ICU nurses and medical practitioners.

This book is not meant to take the place of any textbook on ventilator therapy, but to help as a ready reckoner. The scope of this small book is not anything beyond that.

S Ahanatha Pillai

Acknowledgements

I am extremely grateful to all my teachers, who always made me realise and feel that teaching is a wonderful experience and inspired me to learn that art.

My loving students, both undergraduates and post-graduates consistently inspired me to continue teaching for more than three and a half decades. I am grateful to them for their love to me.

With lot of gratitude, I make special mention about the contribution from my loving wife Mrs Neelam Ahanathan for being a constant source of inspiration and encouragement in all my endeavors, particularly those related to academic ventures and my children for their loving care and the support they give me.

My younger colleagues Prof (Dr) A Paramasivan, MD, DA, Professor and Head of the Department of Anaesthesiology, Government Thoothukudi Medical College, Thoothukudi and Dr G Saravana Kumar, MBBS, DA, helped me in every step of this work. My sincere thanks and love are due to them.

I am immensely grateful to Prof (Dr) E Radhakrishnan, MD, DA, Emeritus Professor, The Tamil Nadu Dr MGR Medical University, Former Professor and Head, Department of Anaesthesiology, Madurai Medical College and Government Rajaji Hospital, Madurai for going through the book and giving a foreword.

My good friend Prof (Dr) J Renganathan, MD, DA, Former Professor and Head, Department of Anaesthesiology and Intensive Respiratory Care Unit, Government Stanley Medical College, Chennai has been kind enough to go through the book and give a foreword. I thank him profusely for that.

I am always very grateful to all my patients for all that they taught me during the past three and a half decades, particularly to those patients treated in the Intensive Respiratory Care Unit, Government Rajaji Hospital, Madurai, as they gave me the

opportunity to learn the clinical applications of ventilator therapy.

My very sincere gratitude is due to Mr R Jayanandan (Senior Author Coordinator) of M/s Jaypee Brothers Medical Publishers (P) Ltd, Chennai, for the excellent and encouraging coordinating work he did with regard to this book as he did with my earlier publications.

I sincerely thank Shri Jitendar P Vij (Chairman and Managing Director) and Mr Tarun Duneja (Director–Publishing) of M/s Jaypee Brothers Medical Publishers (P) Ltd, New Delhi particularly for the inimitable support they rendered in bringing out this book in a very presentable form in a short time.

Contents

CHAPTER 1

Introduction to Mechanical Ventilation

Mechanical ventilation is a mixed blessing as its potential good is not always good enough. While offering hope of prolonged life, mechanical ventilation has drastic implications for the quality of life. Whether a particular individual will benefit from mechanical ventilation is initially a medical judgment. Often, however, no clear diagnosis has been established, and even when one has, the individual's prognosis may remain highly uncertain. The patient, family members, physicians, nurses, and other professional caregivers may not agree with each other on the prognosis and thus, the decision-making reverts from the medical expertise to the realms of psychology, ethics, religion, economics, and law. Furthermore, the costs associated with this technology are enormous. Therefore, for severely ill patients, their families, and those required to make health care decisions, the long-term use of this technology can be the source of considerable anguish.

A patient information series published by the American Thoracic Society gives the following description.

Mechanical ventilation is a life support treatment. A mechanical ventilator is a *machine that helps people breathe when they are not able to breathe enough on their own.* The Mechanical ventilator is also called a Ventilator, Respirator, or Breathing machine. Most patients who need support from a ventilator because of a severe illness are cared for in a hospital's Intensive Care Unit (ICU). People who need a ventilator for a longer time may be in a regular unit of a hospital, a rehabilitation facility, or cared for at home.

WHY ARE VENTILATORS USED?

- To get oxygen into the lungs.
- To get the lungs get rid of carbon dioxide.

- To ease the work of breathing. Some people can breathe, but it is very hard. They feel short of breath and uncomfortable.
- To breathe for a patient who is not breathing because of brain damage or injury (like coma) or high spinal cord injury or very weak muscles.
- If a patient has had a serious injury or illness that causes breathing efforts to stop, *a ventilator can be used to help the lungs breathe until the person recovers.*

This description appears very simple and easy to understand even for a lay man. In clinical practice, it may not be as simple as it sounds, but is a little more.

Many people including some clinicians look at a ventilator with a sense of distaste. Some feel that it is very difficult to understand and manage. The truth is far from that; it is not anything very special; one must realize that a ventilator, after all is a man made machine used worldwide by clinicians, and there should be no difficulty to understand and manage it.

In the past, Anesthesiologists were expected to know how the ventilators work, as many of them were designed on simple mechanical principles. Now, almost all the modern ventilators are microprocessors controlled (computerised) and the technology is so complex that acquiring this extra knowledge is no longer reasonable or justifiable. Now we have the new generation of specialists in every hospital known as Biomedical Engineers and they have the knowledge and maintain the machines.

At this point, we shall recall the following statement of JS Robinson.

"The user must know what the ventilator can do, not how it does that"

—JS Robinson

- Ventilators come in all sizes and shapes to suit many environments and pockets.
- Some are simple bag squeezers and others are versatile microprocessor controlled machines with monitors and alarms.

Not all the time we may be able to procure the most sophisticated ventilator available in the market costing huge money. Nevertheless, we may have a ventilator which can perform the basic functions of a good ventilator. With a good knowledge of applied basic sciences related to respiratory system, probably the available ventilator can be used on vast majority of patients, giving optimum benefits without any problems.

Most of our patients require only the basic modes of ventilation and only very few of them (such as those with chronic respiratory or cardiovascular illness with structural changes in the lung) need sophisticated modes and settings. This simple fact could be realized by younger colleagues in the course of their clinical practice.

Here we may recall the following famous quotation, by Peter Nightingale and J Denis Edwards.

"Unfortunately many Newer Modes have been introduced merely on the basis of Technical Ability rather than as a result of a defined clinical need or demonstrable advantage to the patient."

— *Peter Nightingale and J Denis Edwards*

This statement does not indicate that there is no scope or need for further research in this field and for the development of newer modes of ventilation, but it indicates that available modes can safely be used without grumbling that a better ventilator with the newer modes is not available.

First of all, a thorough knowledge of applied **anatomy** and **physiology** of respiratory system and the **mechanics of normal respiration** is necessary to understand mechanical ventilation and apply it clinically.

There are certain questions to be answered before instituting ventilator support for a patient.

1. What exactly is the requirement of the patient?
2. What exactly the machine which we have with us can do?
3. How best this machine can be used to meet the requirement of the patient?
4. Finally, the most important question is, whether the patient requires ventilatory therapy at all?

Though looking at it superficially, this last question may appear absurd, but long clinical experience has established that ***many a times, making this decision is very difficult and eventually ventilator therapy is instituted in patients not requiring it.*** This fact is infrequently realised in clinical practice, because modern ventilator therapy normally does not do any harm.

If the basic metabolism is reduced due to any reason, eventually O_2 requirement is reduced, CO_2 production is reduced, less metabolite are produced, and the tendency for acidosis is less. Hence minimal reduction in ventilation will not cause hypoxemia and metabolic acidosis as proved by Arterial Blood Gas study.

There are a few more questions which could be answered by an assessment based on *clinical evaluation of ventilation* along with *serial Arterial Blood Gas results*.

- When to put a patient on ventilator?
- What mode of ventilation is needed for the patient?
- When to start weaning the patient?

When someone has inadequate ventilation, some form of support to sustain near normal ventilation without

causing additional damage is quiet sufficient for sustaining and saving the life. If ventilation is maintained for some time even with the crudest method available at hand, time can be bought to get the right type of ventilator needed for that patient.

An event in the history has clearly proved us that even an ordinary resuscitator bag such as Ambu Bag or other such resuscitator bags can be used to maintain ventilation for many days and thus sustain life.

In 1952 in Denmark there was a severe epidemic of paralyzing poliomyelitis. At Blegdam Hospital in Copenhagen, the apparatus available were only one tank ventilator and six cuirass ventilators, but the number of patients requiring respiratory assistance was very high. By this time "controlled respiration" was well established in anesthesia and as a last resort this technique was extended to patients needing long-term ventilation. At one time 70 patients were receiving respiratory assistance from "ventilators which consisted of medical students squeezing the bag of a *to and fro* system with carbon dioxide absorption". Medical students were made to do this in a shift of 8 hours duty and were paid for each shift. More than 30 patients survived.

Mostly, improper management in ventilator therapy is likely to be caused by any of the following reasons.

- Improper assessment of the patient's condition.
- Inability to decide about the patient's requirement for ventilatory support.
- Inadequate knowledge about the ventilator settings (Not the mechanism by which it ventilates the lung).
- Not knowing the limitations of the ventilator available, to do the job which we expect.

It is quite obvious that such a therapy will result in serious complications.

At this point, there may be a need for a question from the reader; "Will I be able to operate all ventilators by reading this book?" The straight forward, simple and honest answer is – "It may not be possible immediately".

However, the descriptions in this book will give a very clear idea as to how the basic sciences could be usefully applied to a patient on ventilator. That is, with the orientation of the *mechanism of normal respiration* and the *mechanism of artificial respiration* in mind, applying artificial ventilation for the patient. Then make the necessary modulations in that, to achieve the best form of ventilation (the best suited mode) for the particular patient. Certainly everything else can be built on that basis.

The principle involved in their use must be understood. If an unfamiliar ventilator is encountered, for the first time, we will certainly be worried how to operate it? It can be done by any one of the following methods.

- The manufacturer's "User hand book" must be used. *Carefully read the operating manual fully. Then connect a dummy lung (a rubber bag meant for that purpose) to the ventilator and try all the modes and settings in that to understand it well.* It is always helpful to use a "dummy lung" and understand the "Capabilities" and "Limitations" of a particular ventilator.
- Getting the relevant information directly from some one who is using the particular ventilator routinely. *He can explain briefly the operating modalities; modes and settings, and the method of operating it. He can even operate it and explain all about it.* It will be an easier short cut method of knowing about it.

It is potentially hazardous to connect the patient to an unfamiliar ventilator and attempting to set the mode and other settings. As the patient who needs ventilator support

is usually a critically ill person, he may not stand even minimal insults in this process.

In such a situation, the patient can be manually ventilated by connecting him to an Ambu Bag or to an anesthetic machine. In the meanwhile a dummy lung can be connected to the ventilator and operated to know clearly about the settings and then the ventilator with proper settings needed may be connected to the patient.

Whatever way it is done, we have to be concerned about four main aspects.

1.	Volume of ventilation:	It must be adequate. Not more.
2.	Mean airway pressure:	It must be optimal.
3.	Distribution of gases in lung:	It must be uniform to all areas.
4.	Diffusion of gases:	It must be adequate. If not adequate, FRC has to be slightly raised so that more alveoli are recruited to take part in the diffusion.

For that purpose, the knowledge of applied aspects of the following is essential and is discussed in the preliminary chapters.

- *Respiratory Anatomy*
- *Respiratory Physiology*
- *Respiratory Mechanics*
- *Mechanics of Artificial Respiration (Mechanical ventilation).*

The sophistications if needed can be added one by one in the ventilatory support after carefully studying the actual requirement of the patient and also based on his improvement after starting the therapy.

In a critically ill person, once ventilatory therapy is instituted, the normal physiological range of pressures, both **"intrapulmonary"** and **"intrapleural"** may be modified causing significant hemodynamic compromise

and in turn may result in serious cardiovascular collapse. This is likely to be more severe, if the patient has depressed cardiovascular reflexes either due to autonomic imbalance or due to severe depression of central nervous system.

When the ventilator therapy is instituted, the means for managing such an eventuality if occurs must be at hand.

The most important aspect of respiration is that in normal conditions of life it happens by the control of the respiratory center, modulated by various reflexes related to the regulation of respiration. *It is so effortless that obviously no one normally realizes that breathing is going on.* Only when there is a problem, respiration is realized as a work and that increased work of breathing is unpleasant and distressing. It is abnormal (pathological), and there we may need a ventilator to support respiration.

With this information of introduction we may have further discussions about the ventilator therapy to understand it in the simplest way possible.

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CHAPTER 2

History of Mechanical Ventilation

'The lungs are the center of the universe
and the seat of the soul'
—Unknown Philosopher

It will be interesting to go into the history, to know how and when the artificial respiration was started and how and when the developments were added on to it to the extent of having the modern sophisticated microprocessor controlled ventilators today.

Mechanical ventilation is the mainstay of Resuscitation, Intensive Care Medicine, and Anesthesia. ***Mechanical ventilation as with many things in medicine was born out of necessity.***

- For centuries, it has been realised that failure to breathe need not necessarily lead to death.
- *The first report of successful artificial respiration is to be found in the Holy Bible*. In Old Testament (800BC), in the Second Book of Kings, there is a graphic description of resuscitation of an apparently dead child by the Prophet Elisha. Prophet Elisha induced pressure breathing from his mouth into the mouth of a child who was dying (Kings 4: 34–35).
  It reads as follows;
  "And he went up and lay upon the child, and put his mouth upon his mouth, and his eyes upon his eyes, and his hands upon his hands ……. And he stretched himself upon the child, and the flesh of the child waxed warm …….. and the child sneezed seven times ..…… and the child opened his eyes."
- Hippocrates (460–75 BC) wrote in his book – *"Treatise on Air""*– 'One should introduce a cannula into the trachea along the jaw bone so that air can be drawn into the lungs'. This is the first written instance of *Endo-tracheal Intubation.*

- Paracelsus (1493–1541) used 'Fire Bellows' connected to a tube inserted into patient's mouth as a device for assisted ventilation. This was the first study (1550) which credited him of the first form of *Mechanical Ventilation.*
- In the end of 18th century in 1767, "The Society for the Recovery of Drowned Persons" was formed in Amsterdam.
- The first successful human resuscitation by mouth to mouth breathing was described by John Fothergill in England in 1774.
- In 1774, Dr. William Haves, John Hunter and thirty others founded the "Humane Society" which was later renamed as "Royal Humane Society".

Medals and prizes were awarded for new ideas and new apparatus for resuscitation and many types of equipment based on pumps, bellows, and tubes were introduced and became popular. The idea suggested by Vesalius and Paracelsus 200 years previously (use of "Fire Bellows" suggested in 1541) was brought into use again.

- Unfortunately, the danger of very high airway pressure was not realised and it lead to many deaths due to ***Tension Pneumothorax.*** In 1827, in French Academy of Science, Leroy demonstrated that it is possible to rupture the alveolus by high airway pressure. He opined, ***"Many patients who would otherwise have recovered were speedily dispatched by overenthusiastic use of bellow equipments."***
- In 1829, Magendie confirmed Leroy's findings and the use of bellows for ventilating the lungs fell into disrepute.
- In 1837, The Royal Humane Society recommended manual compression of chest if artificial ventilation was necessary.

- Ventilation with bellows slowly reappeared sporadically in the next 100 years, but regained wide acceptance when Kreiselman introduced his apparatus during Second World War.
- The Ruben's self-refilling bag was a later development on the same principle.
- In 1743, Tossach advocated the mouth to mouth resuscitation done by Prophet Elisha, but this method was later condemned as unhygienic (practically true) by the newly founded Royal Humana Society.
- Mouth to mouth breathing was shown to be physiologically correct by Elam in 1954.
- In 1958, Peter Safar demonstrated that the ventilation provided by mouth to mouth breathing was superior to that obtained by manual chest compression and arm lift maneuvers. (Sylvester's method). Now, ***expired air ventilation*** (mouth to mouth breathing) is regarded as the method of choice when equipments are not available.
- During the 100 years from 1840 to 1940, most of the mechanical aids for artificial ventilation depended on applying a subatmospheric pressure (negative pressure) outside the thorax.
- Dr Alfred F Jones of Lexington, Kentucky, patented the first American tank ventilator in 1864. The interesting and funny part of this invention is that, he claimed this as a cure for "paralysis, neuralgia, rheumatism, seminal weakness, bronchitis, dyspepsia, and many other diseases including deafness" but the inventor of subsequent tank and cuirass ventilator claimed only successful treatment of respiratory diseases.
- Alexander Graham Bell devised a vacuum jacket for resuscitation of new born in 1889. Dr Egon Braun of Boston described a small tank ventilator for new born.

- In 1920, gas and electricity supply industries, dissatisfied with the available methods for treating victims of electric shock and gas poisoning, requested Phillip Drinker of Harvard for advice on resuscitation.
- In 1929, Philip Drinker and his colleagues introduced their tank ventilator for prolonged artificial respiration which subsequently became known as the **"Iron lung"** (Fig. 2.4).
- Poliomyelitis was the common cause of respiratory failure in children and young adults during the first half of that century, and *the first patient to be treated on the "Iron lung' was a victim of paralytic poliomyelitis* (Figs 2.2 and 2.3). Modifications in the basic design were made over the next 25 years (Figs 2.4 to 2.9).
- In 1950, Ray Bennet and colleagues developed a method of supplementing intermittent positive pressure synchronizing with the negative pressure ventilation and made the attachments for that.
- Further, development of cuirass ventilator proved useful during the recovery from the acute phase of the disease.
- Negative pressure ventilators (Fig. 2.10) were in extensive use during the polioepidemic in Los Angeles in 1948 and that in Scandinavia in 1952.
- In 1952 in Denmark there was a severe epidemic of paralyzing poliomyelitis. At Blegdam Hospital in Copenhagen, the apparatus available were only one tank ventilator and six cuirass ventilators, but the number of patients requiring respiratory assistance was very high. By this time, "controlled respiration" was well established in anesthesia and as a last resort; this technique was extended to patients needing long-term ventilation. At one time, 70 patients were receiving respiratory assistance from "ventilators which consisted of medical students squeezing the bag of a

to and fro system with carbon dioxide absorption". Medical students were made to do this in a shift of 8 hours duty and were paid for each shift. More than 30 patients survived.

- Lassen and Ibsen established the basic principles of long-term ventilation: *Careful airway control and protection, humidification, avoidance of high inspired oxygen concentration and meticulous physiotherapy.*
- Once the acute phase was over, weaning was accomplished by a forerunner of intermittent mandatory ventilation (IMV)
- *A dramatic fall in mortality occurred after the new technique had been introduced, and this ensured that intermittent positive pressure ventilation (IPPV) was to become the standard method of artificial ventilation.*
- The superiority of IPPV was confirmed during the Stockholm epidemic in the following year and during the New England epidemic of 1955.
- After the introduction of the Salk and Sabin vaccines, the incidence of poliomyelitis fell sharply. However, the skills which had been developed were put to good use in the ventilatory management of polyneuritis, drug over dosage and chest trauma.
- During the 1960s, the indications for IPPV were broadened and the cardiovascular effects were investigated.
- Improvement in the immediate management of the injured showed that a group of patients who had been successfully resuscitated developed a lung condition characterized by certain radiological changes and severe impairment of gas exchange. "Shock lung" and "Adult Respiratory Distress Syndrome" (ARDS) are the two of the many names for the condition.

- Positive end expiratory pressure (PEEP) was added to IPPV to support the ARDS patients to have better gas exchange, but the recognition of adverse effects of PEEP on the circulation started the quest for an "ideal" PEEP level that would balance the respiratory advantage over the circulatory disadvantage.
- In spite of better understanding of ARDS, the mortality rate of this condition remained disappointingly high.
- In 1970s, extracorporeal technique of oxygenation was tried with a little good result for long-term ventilation.
- A few centers experimented with High Frequency Ventilation (HFV), although the true role of this interesting technique in long-term ventilation is yet to be established.
- The benefits of long-term ventilation have not always been universally accepted. In the 1960s, the term *"respirator lung"* was introduced to describe radiological and pathological changes in the lungs of some patients who had received artificial ventilation. In retrospect, pulmonary **oxygen toxicity** was probably responsible for many cases of "respirator lung".
- The toxic effects of pure oxygen on lung at standard atmospheric pressure had been recognized for over 50 years and inspired oxygen concentrations of 50% or less was considered safe.
- After 1955, most ventilators in North America were powered by compressed oxygen and employed a venturi device to entrain air. It was erroneously assumed that these ventilators would deliver gas where oxygen concentration was below 50%. Some years later, it was found that the venturi device was not performing as expected. As a result, oxygen concentration in the toxic range was being delivered to the patients.

- This evidence, together with the experience already gained with polio patients, established that IPPV with modest levels of oxygen in the inspired gas may be continued indefinitely without adverse effects on lung.
- Some still view long-term artificial ventilation with deep suspicion even though its value in intensive care is now established beyond doubt.
- After the Second World War, there were two stimuli to the development of mechanical ventilator for IPPV. *The first was the introduction of Curare into anesthesia. The second was the fear of health authorities that if another epidemic of poliomyelitis occurred, large number of patients might require artificial ventilation.*

TANK VENTILATORS AND CUIRASS VENTILATORS

- These equipments once used extensively almost for a century have now become extinct and are of historical value. However, the enthusiasm of the inventors in developing such large and complicated equipments for supporting the ventilation of patients with respiratory failure has to be admired and appreciated with respect.
- These machines ventilate the patient by applying a sub-atmospheric pressure to the outside of the thorax. This often called as **Intermittent Negative Pressure Ventilation** (INPV).
- The first tank ventilator was introduced in 1832 and the first Cuirass in 1874.

GENERAL DESCRIPTION

- The patient is kept on a mattress in an airtight cabinet from which only his head protrudes outside. A padded collar round the neck forms an effective seal (Fig. 2.1A).

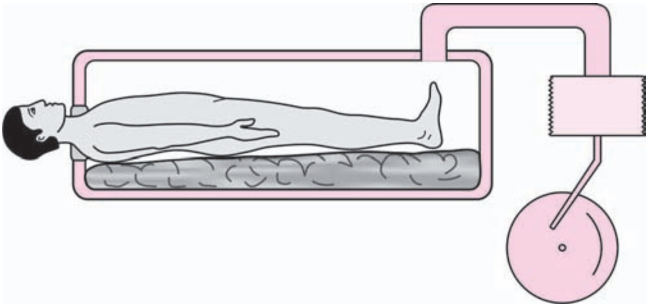

Fig. 2.1A: Principle of negative pressure ventilator

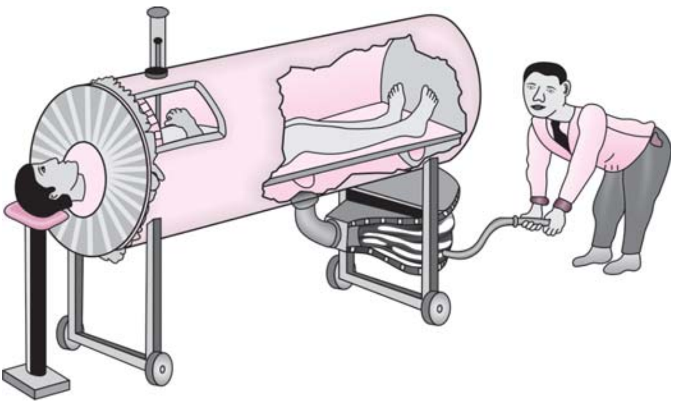

Fig. 2.1B: A primitive tank ventilator with bellows operated manually

- The pressure inside the cabinet is lowered rhythmically by a system of pumps or a set of bellows and then allowed to return to normal atmospheric level (Fig. 2.1B).
- The first power driven tank ventilator, the **"Iron lung"** was developed by Drinker, McKhann and Shaw in 1929.

Fig. 2.2: An iron lung ward filled with polio patients, Rancho Los Amigos Hospital, 1953

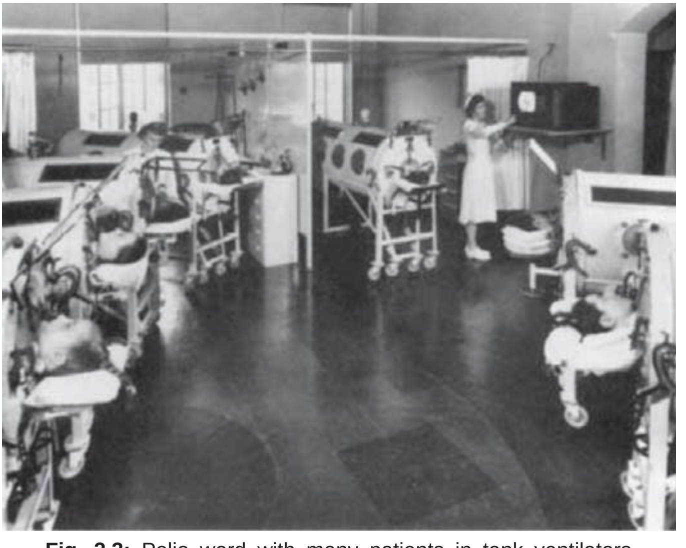

Fig. 2.3: Polio ward with many patients in tank ventilators

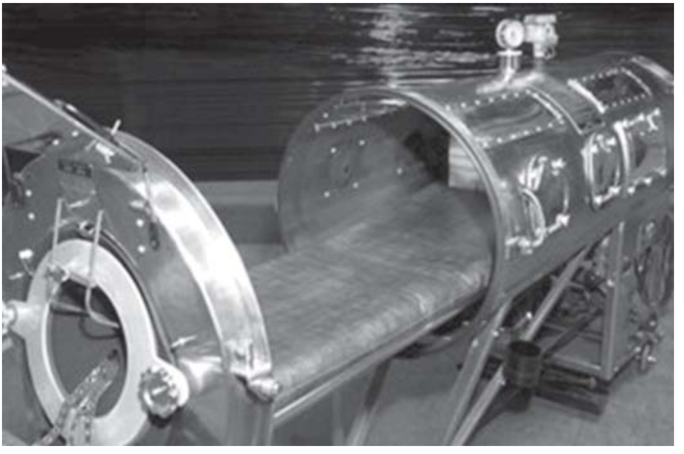

Fig. 2.4: An emerson iron lung

- ❖ The patient lies within the chamber which when sealed provide an effective oscillating atmospheric pressure.
- ❖ This particular machine was donated to the “Centers for Disease Control and Prevention” Museum by the family of a Polio patient, Barton Hebert of Covington, Louisiana, who had used this device from late 1950s until his death in 2003.

- There were port-holes in the side of the tank through which the patient could be observed and sealed ports to allow the use of manometers, blood pressure cuffs, and stethoscopes.
- Many modifications of the original design were made over the next 25 years to make the patient more accessible and to add a “positive” phase in the tank to assist expiration.
- The Kelleher Rotating Tank Ventilator has the provision to rotate the tank in its long axis, so that the back of the patient’s chest become accessible for auscultation and physiotherapy.

Fig. 2.5: Blue iron lung

- This Blue Iron Lung is the first one made by John Emerson's Company.
- He tested it by spending the night in it.
- It was first used in Providence, Rhodi Islands, in 1931 to save the life of a priest suffering polio.

- The two main disadvantages of tank ventilators are that the access to the patient for nursing care and physiotherapy is restricted, and that the airway is not usually protected. Vomiting and regurgitation are particularly hazardous during INPV, even for patients with normal pharyngeal and laryngeal reflexes. If vomiting occurs, a port should be opened immediately to equalize the pressure inside and outside of the tank and so to reduce the risk of aspiration.
- Apart from these the negative pressure applied over the whole body restricts the venous return to the heart.

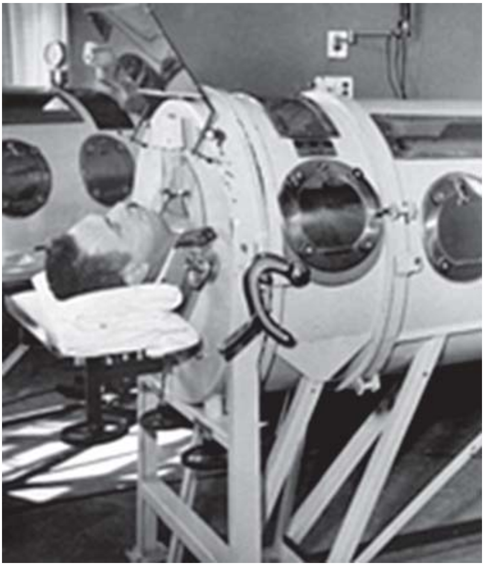

Fig. 2.6: A man using an Emerson tank respirator equipped with a mirror

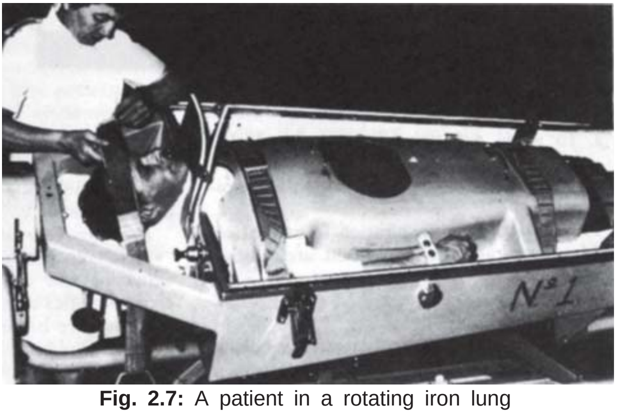

Fig. 2.7: A patient in a rotating iron lung

- ❖ This can be tilted in its long axis for examining and nursing the patient.

Fig. 2.8: A different model of iron lung with the motor and bellows at the bottom

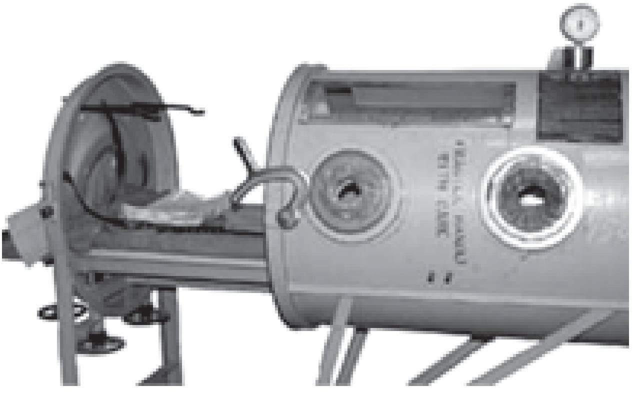

Fig. 2.9: The iron lung in open condition

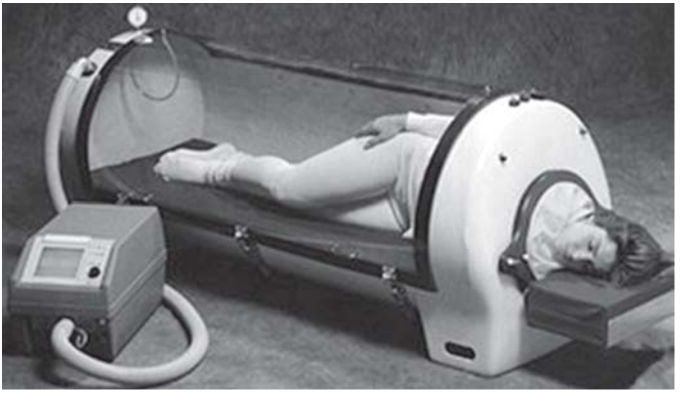

Fig. 2.10: Patient in the tank of a modern negative pressure ventilator

- ❖ The tank has clear acrylic lid and a gasket around the patient's neck
- ❖ The ventilator machine itself is seen in the front as small box.

DESCRIPTION OF A CUIRASS VENTILATOR

- Cuirass ventilators are named after 15th century body armour which consisted of a breastplate and back plate fastened together.
- A rigid shell fits over the thorax and upper abdomen and the padded rim makes contact with the skin to form an airtight seal (Fig. 2.11A).
- A bellows is connected by flexible tubing to the air space between the skin and the shell; expansion of the bellows creates a sub-atmospheric pressure in the air space during inspiration (Figs 2.11B, 2.12 and 2.13).
- Cuirass ventilators leave patient's arm and legs free and cause less circulatory embarrassment than tank ventilators (Fig. 2.14).
- They are less efficient than tank ventilators and the tidal volume obtained by a given subatmospheric pressure is smaller.

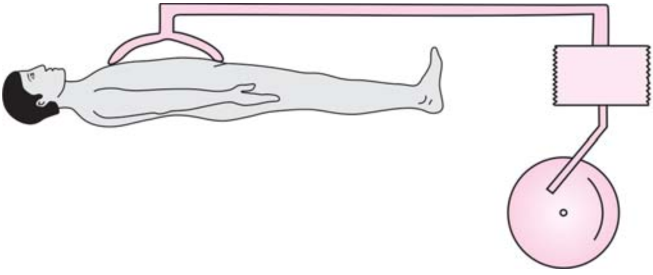

Fig. 2.11A: Principle of cuirass ventilator

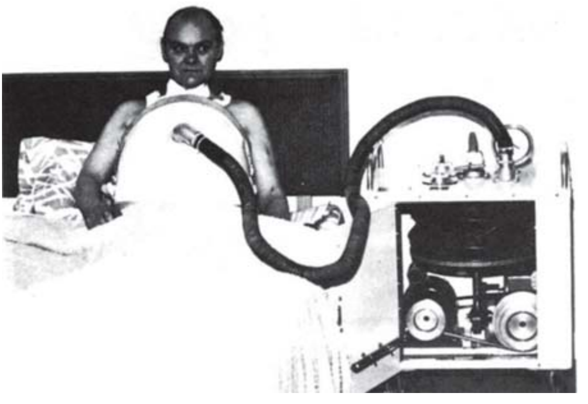

Fig. 2.11B: The cuirass ventilator with the bellows and the motor

- The larger the cuirass shell, the more the performance approaches that of the tank ventilator.
- Cuirass ventilators were used as assisters for patients who have chronic respiratory impairment or those who are recovering from some episode of paralysis.
- Particularly the patients who develop respiratory insufficiency during sleep were well supported with this device.

Fig. 2.12: A patient being supported with a cuirass

Fig. 2.13: A modern cuirass with its motor, bellows and controls

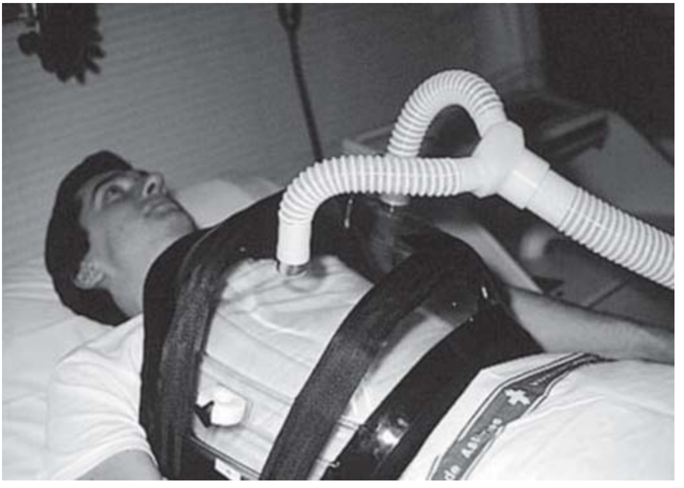

Fig. 2.14: A patient (Model) showing the way the cuirass is applied

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CHAPTER 3

Anatomy of Respiratory System

- *Anatomy of airway: Upper airway and lower airway*
- *Upper airway: Nasal passages: Sinuses; Pharynx; Larynx*
- *Lower airway: Trachea, conducting airways (nonalveolate region)*
- *Respiratory zone (Alveolate region)*
- *Nerve supply to respiratory system*
- *Blood supply to respiratory system*
- *Lymphatic drainage*
- *Thoracic cage*

Adequate knowledge of applied anatomy and physiology is essential for instituing ventilator support (Artificial Respiration).

While discussing applied functional anatomy, inevitably some physiological aspects have to be included, because every anatomical basis is for maintaining a normal physiological function.

The respiration is a remarkable vital function controlled very precisely and perfectly by the anatomical and physiological mechanisms that *any one under normal conditions, ever realises that he is breathing with such a degree of regularity and precision.* Still we have a certain degree of voluntary control on respiration for a short time. In that way, it is remarkable and complicated. Only when there is any problem with any part of the system, to the extent of making the respiration inadequate, we realise that this vital function was going on well, so long, perfectly.

The overall function of this system is to provide life sustaining oxygen to all the cells of the body and to remove the byproduct of cellular metabolism, carbon dioxide. By this way, an efficient pulmonary system together with cardiovascular system is intensely related to the metabolic process of the body. Therefore, the knowledge of pulmonary and

respiratory anatomy provides a sound foundation for understanding the complex process of respiration.

For making a useful discussion, the anatomy of respiratory system can be broadly divided into two;

- The anatomy of air passages and alveoli where gaseous exchange occurs (anatomy of *airway and diffusion*).
- The anatomy of the thoracic cage and the muscles of respiration (anatomy related to the *mechanics of respiration*).

(This part *"anatomy of thoracic cage and muscles of respiration"* will be dealt with separately in the chapter on *"Mechanics of Respiration".)*

In this chapter, we shall discuss the anatomy related to the conducting airway and that related to diffusion of gases (Alveolar zone).

ANATOMY OF AIRWAY

For the convenience of discussion, the airway may be divided into ***Upper Airway*** and ***Lower Airway.***

Upper Airway

This consists of the following components.

1. Nasal passages
2. Sinuses
3. Pharynx
4. Epiglottis
5. Larynx

The three important functions of the upper airway in general are:

- Conducting the air to the lower airway.
- Protecting the lower airway from foreign matter such as food or liquids soiling it.

- Filtering, warming, and humidifying the inspired air for efficient gas exchange.

Nasal Passages

When a patient is to be mechanically ventilated, passing an endotracheal tube into the trachea (*endotracheal intubation*) is mandatory for the purpose of securing the airway. Incidentally it is an ideal and the best airway for ventilating the lungs. This artificial airway (whether passed via nose or mouth) inevitably bypasses the normal airway of nasal passages.

During normal respiration, the nasal passages perform the most important functions of *filtering, humidifying, and warming the air and more*.

All these functions of nose, essential for the normal functioning of the respiratory system will be lost in an intubated patient. Hence, to protect the respiratory tract from damages, suitable arrangements are to be made for *filtering, humidifying, warming the air* during the process of mechanical ventilation, to adequately compensate for the absence of the natural protection.

The applied functional anatomy of nasal passages may be discussed briefly below.

- The two nasal cavities start in front from the external nares and end posteriorly in the nasopharynx.
- The structure of the nose, with its two nasal cavities, turbinates, and rich vasculature provide maximum contact between the inspired air and the nasal mucosa for ***humidification of the air.***
- The stiff hairs in the anterior part of the nasal fossa, together with the spongy mucous membrane and the ciliated epithelium comprise a powerful ***defence against invasion of any organism.***

- In reserve are the flushing action of the watery secretions and the *bactericidal properties of these secretions.*
- Convolutions of cartilaginous tissue known as turbinates provide an increased surface area for *warming, humidifying and filtering of inspired air*.
- ***Warming and humidifying the inspired air is probably the most important work of nose.*** The magnitude of the task can be realised only when we recall that nearly 10,000 liters of air pass through nose every 24 hours.
- The great vascularity of mucosa helps to maintain a constant temperature of the air reaching the alveoli. The inspired air, which is cold, at about 20°C is warmed to approximately 37°C during its passage through the nose and variations in external temperature ranging from 25°–30°C produce less than 1°C change in the temperature of air reaching the laryngeal inlet.
- Temperature adjustment and humidification begins as soon as the air enters the anterior nasal cavity; by the time inspired air reaches the alveoli, it is 100% saturated with water vapour.
- The supply of moisture comes partly from transudations of fluid through the mucosal epithelium and to a less extent from the secretions of glands and goblet cells in the nasal mucous membrane. The daily volume of nasal secretions is about 1 litre, of which about three quarters (about 750 ml) is utilised for saturating the inspired air.
- The optimum relative humidity of room air is only about 45 to 55 % but the *bronchi and alveoli require 95% humidity for adequate function.*
- The nasal cilia which are very fine microscopic hair like projections sway together in waves to move the trapped particles posteriorly.

- ***Throughout the respiratory tract, the continuous activity of cilia is probably the most important single factor in the prevention of the accumulation of secretions.*** In the nose, material is swept towards the pharynx and in the bronchial tree; the flow is carried towards the entrance of larynx. ***The absence of moisture, even for a few minutes, leads to cessation of ciliary activity.***
- The cilia cannot work without a blanket of mucus. Together it is called as *"Mucociliary Escalator"*. The ciliary activity will be lost if the mucus layer is dried by breathing dry gas for long time, or if the temperature of mucosa falls to 7 to 10°C.
- The blood supply to the nasal mucosa is controlled by an elaborate autonomic reflex which enables the mucosa to swell or shrink on demand.
- There is a dual nerve supply to this area. *Parasympathetic* fibers pass via the facial, greater superficial petrosal, and vidian nerves to relay in the sphenopalatine ganglion.
- *Sympathetic* fibers reach the ganglion from the plexus surrounding internal carotid artery via the vidian nerve.
- *Sensory* nerve supply to the nasal mucosa come from the first and second division of trigeminal nerve. Anterior half of the septum and lateral wall are supplied by the anterior ethmoidal branch of nasociliary nerve (first division) and the posterior two thirds by the nasopalatine nerves through sphenopalatine ganglion.

It has to be reemphasized and remembered here that, when an artificial airway (Endotracheal Tube or Tracheostomy Tube) is in place, these functions of warming, filtering and humidification of inspired air, are bypassed and must be provided for the patient.

In addition to that, the *mucociliary escalator stops* at the level of the tip of the endotracheal tube or tracheostomy

tube, causing collection of secretions at that level resulting in encrustation and blocking of the tube.

Sinuses

Similarly, the paranasal and the other sinuses play very important role in modifying the quality of the air breathed during normal respiration. Their applied anatomy is discussed briefly below.

- Sinuses are air spaces that decrease the weight of the skull, *provide mucous for the nasal cavity*, and are important as resonance chambers of voice.
- The paranasal sinuses are important and include frontal, ethmoidal, sphenoidal, and maxillary sinuses. All are lined with *ciliated mucous producing cells* and have small pathways known as meatus that communicate with the nasal cavity, lying underneath the nasal turbinates.
- *Occlusion of the meatus causes fluid to accumulate in the sinuses leading on to infection-sinusitis.* ***The sinus infection is common problem encountered in patients on prolonged ventilator therapy in general and more so with nasotracheal intubations.***

Pharynx

Pharynx is actually a common passage or junction where air and food pass through to take their respective route. The applied aspect of anatomy of this part of upper airway is that, *it is this area which gets obstructed when the patient becomes unconscious*. Let us discuss that briefly here.

- Air from the nasal cavity enters into the space behind the nasal cavity and oral cavity called the pharynx.

- The pharynx extends from the posterior aspect of the nose at the base of skull down to the lower border of the cricoid cartilage where it becomes continuous with esophagus, and the respiratory tract through the larynx.
- There are three divisions of pharynx; the **nasopharynx**, the **oropharynx**, and the **laryngeal pharynx** or **hypopharynx** (Fig. 3.1).

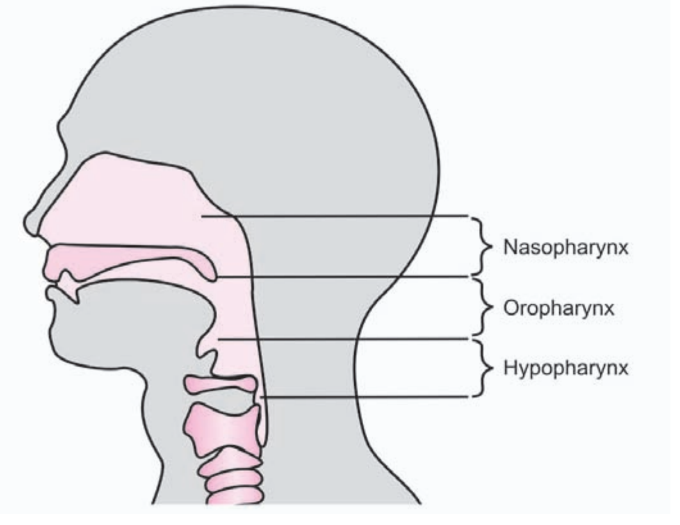


Fig. 3.1: Pharynx and its three divisions

- The nasopharynx begins at the base of the nasal cavities and extends to the soft palate. It contains eustachian tubes and the lymphoid tissue 'adenoids'.
- The oropharynx extends from the soft palate and uvula to the epiglottis. It is visible with the mouth open and the tongue depressed.

- The laryngopharynx or hypopharynx contains the larynx and it is the critical dividing point of solids and liquids from air. *In fact, when Laryngeal Mask Airway is placed in the right position, tip of the LMA will be in the hypopharynx, so that the mask aperture correctly faces the laryngeal inlet.*
- The principal difficulty in maintaining a perfect airway in an unconscious patient is the tendency of the tongue to fall backwards (sagging backwards and the base of tongue sitting on the posterior pharyngeal wall) and obstruct the laryngeal opening. This occurs as soon as the consciousness is lost and the muscles supporting the tongue start to relax (Fig. 3.2).

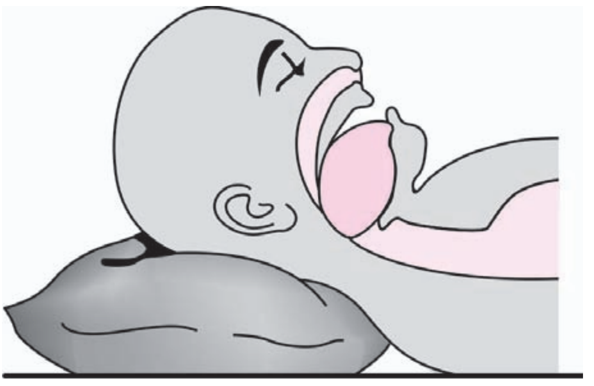


Fig. 3.2: Common mechanism of airway obstruction in an unconscious patient

❖ The mandible is relaxed.
❖ The neck is flexed.
❖ Total airway obstruction by the tongue sitting on the posterior pharyngeal wall.

- To obtain a clear airway, two separate maneuvers are required to provide perfect airway in the unconscious

patient. First the lower jaw must be carried forward and upwards so that the lower incisor teeth lie in front of the upper incisor; known as the 'Prognathic attitude'. In the second maneuver, the head is hyper extended so that the tongue is carried farther upward and forward, away from the posterior pharyngeal wall (Fig. 3.3).

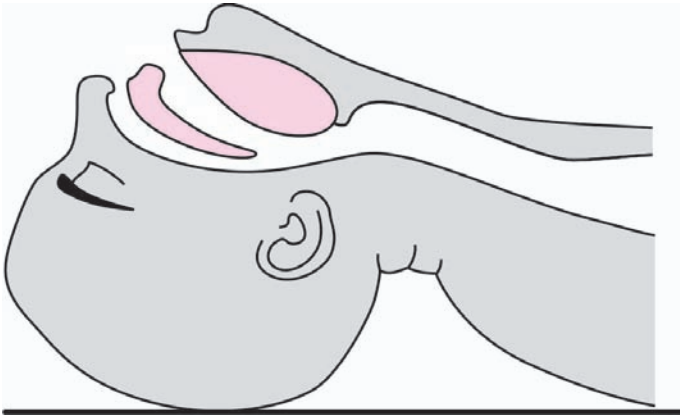


Fig. 3.3: The airway obstruction relieved
- Extension of the head.
- Pushing the mandible upwards (Prognathic attitude).

Larynx

Originally in lower animals the larynx was a protective sphincter valve for the respiratory tract and later through evolution in human beings it became an organ of phonation also. In adults, the space in between the vocal cords (glottis) is the narrowest portion in the upper airway and the endotracheal tube passes through this opening only.

The clinical significance is that the biggest tube that can be passed through this opening without difficulty

(optimum size) must be used to keep the resistance to breathing minimal. Too small tube will cause serious increase in resistance to respiration whereas too big a tube may cause severe reflex stimulation and post-extubation oedema and stridor. The applied anatomy is discussed below.

- The larynx lies at the levels of the 3rd to 6th cervical vertebrae and comprises of a number of articulated cartilages surrounding the upper end of trachea.
- The larynx contains the vocal cords for phonation and is also an organ with sphincter functions that prevents aspiration.
- The principal cartilages of the larynx are the thyroid, arytenoids, and the cricoid. The largest and the most superior of the cartilage is the thyroid (meaning, "Shield like). Incidentally, it protects the delicate structures of larynx from any damage from front.
- The cricoid cartilage lies just below the thyroid and is attached to it by the cricothyroid membrane. *It is this membrane that is incised to perform an emergency procedure,* ***the cricothyroidotomy****, for upper airway obstruction* (Fig. 3.4).
- The arytenoid cartilage serves as attachment for vocal cord ligaments. It swings in and out from a fixed point, thus opening and closing the space between the vocal cords. The vocal cords are drawn apart during inspiration and relax towards midline during expiration. *It is the reason for advising to extubate a patient during inspiration when the vocal cords are wide apart and less damage to vocal cords are caused.*
- The glottis is the space between the vocal cords.
- The epiglottis is the leaf like cartilaginous structure extending from the base of the tongue and attached to the thyroid cartilage by ligaments. It projects upward

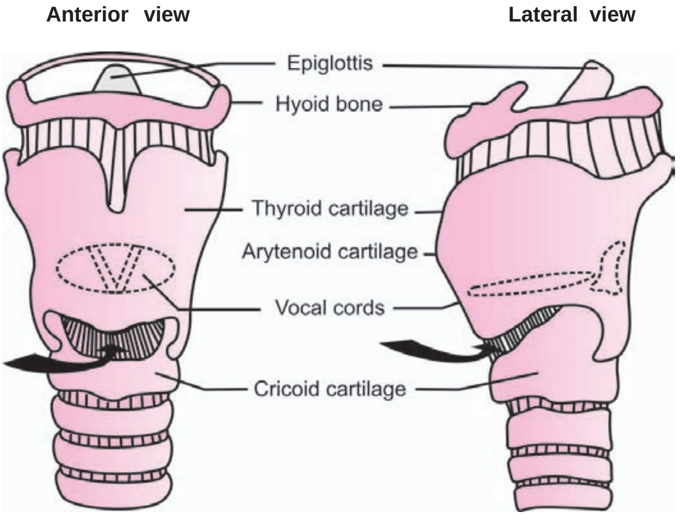


Fig. 3.4: Cartilages of larynx and upper end of trachea (The arrow shows the cricothyroid membrane where emergency cricothyroidotomy will be done)

and posteriorly. During swallowing the epiglottis flaps down to direct the swallowed material into the esophagus, thus guarding the opening of the larynx.

Lower Airway

Lower airway consists of the trachea which is the downward continuation of the larynx, which divides into the **right** and **left** main bronchus leading to the right and left lungs. The main bronchus divides into the branches for the individual lobes of the lungs. The **lobar bronchi** further divide into **lobular bronchi** and finally divide into smaller tubes like the branches of a tree becoming smaller and shorter.

Its functions are;

- Conducting air lower down to alveoli
- Providing mucociliary defence

Trachea

Applied anatomy of trachea is essential when an attempt is made intubate and ventilate a patient, as the endotracheal tube is to be placed in the proximal part of the trachea. It is necessary to see that the endotracheal tube is not advanced too much into the trachea.

The tube may touch the carina (the area of bifurcation of trachea) and irritate, to cause severe reflexes or it may be introduced into a main bronchus to cause one lung (Unilateral) ventilation. Usually, because the right main bronchus is in line with the trachea, right sided intubation is common (Figs 3.5 and 3.6).

- Trachea is a tube formed of rings of cartilages which are incomplete posteriorly. The posterior portion of trachea is made of smooth muscle and lies adjacent to the esophagus. *Excessive pressure on this smooth muscle by the cuff of an artificial airway (Endotracheal tube or tracheostomy tube) can lead to erosion and tracheo-esophageal fistula.*
- It is about 10–11 cm long, extending downwards from the lower part of larynx opposite the level of 6th cervical vertebra to the point of its bifurcation into the right and left main bronchus at the carina, about the upper border of 5th thoracic vertebra. Anteriorly, it corresponds with the junction of the body of sternum and manubrium sterni, the angle of Louis.
- In children, the carina (the level of bifurcation) is on a level with 3rd costal cartilage.
- The diameter of trachea is about 1.2 cm, much smaller in a child, and about 3 mm in a neonate.

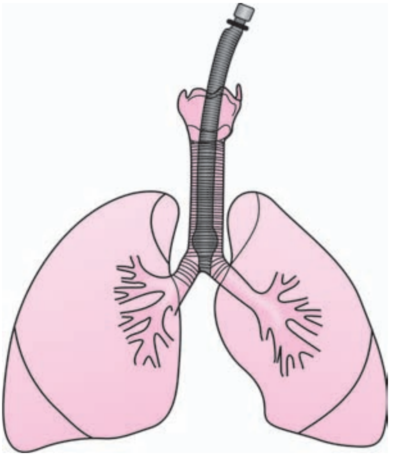


Fig. 3.5: Endotracheal tube introduced too far in trachea touching carina

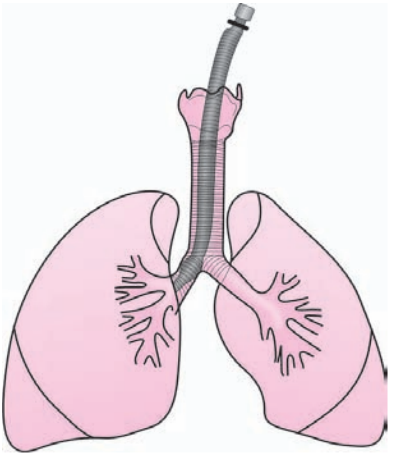


Fig. 3.6: Endotracheal tube in right main bronchus

- Blood supply is from two sources; upper two thirds supplied by *inferior thyroid artery* and the lower one third by *the bronchial arteries*. The arteries run circumferentially with few anastomoses in the long axis of trachea.
- It is lined by ciliated columnar epithelium and mucous secreting goblet cells.
- The trachea moves with respiration and changes in position with the movement of the head. During deep inspiration, the carina (the point of bifurcation of trachea) can descend as much as 2.5 cm. Similarly the extension of the head and neck, the ideal position for maintaining the airway in an unconscious patient, can increase the length of trachea by as much as 23–30%.
- *Clinically, if a patient is intubated with the head in flexed position at atlanto-occipital joint, and the endotracheal tube is too short that it just reaches beyond the vocal cords, the subsequent hyperextension of the head may withdraw the tube into the pharynx.*
- Tracheostomy is always performed at a level below the first tracheal ring cartilage, as section of this structure may later lead to the development of stricture. Ideally it is done below the second tracheal ring.
- One of the principal problems following tracheostomy is the prevention of drying and encrustation of the mucous membrane in the trachea and main bronchi. This may rapidly develop despite humidification of the air.
- The trachea divides into the two main stem bronchi (main bronchi). The main stem bronchi consist of circumferential *smooth muscle and plates of cartilage* that irregularly encircle the airway. The smooth muscle constricts in response to certain stimuli. The lining of the bronchi are *ciliated epithelium* and *more mucous*

producing goblet cells. Progressing distally, in the airways there is loss of cartilage, mucous secreting cells, and cilia.

Lower airway is divided into two based on their function.

1. **Conducting airways:** Nonalveolate region.
2. **Respiratory zone:** Alveolate region.

Conducting Airways (Nonalveolate Region)

- As it is discussed, the trachea divides into two main bronchi, right and left.

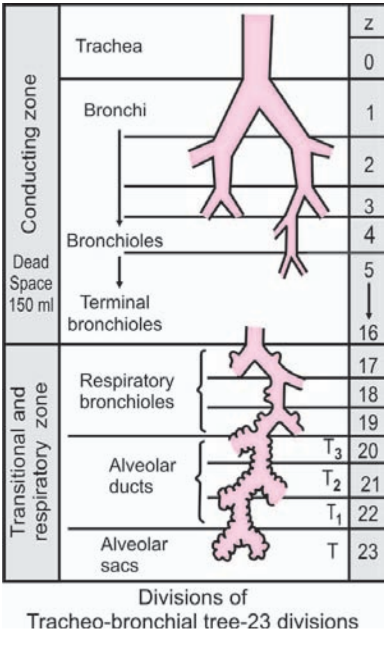


Fig. 3.7A

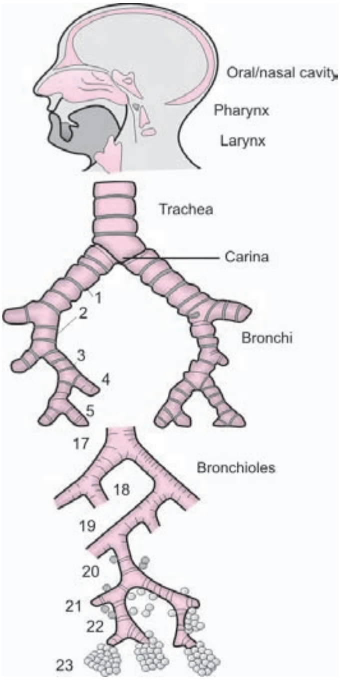


Fig. 3.7B

Figs 3.7A and B: 23 divisions of tracheobronchial tree

- Taking **the main bronchus** as **division 1** subsequent divisions are numbered as 2, 3 and so on. The divisions are otherwise called as **generation.**
- Approximately the first 16 divisions of the tracheobronchial tree take no direct part in gas exchange and are designated as the conduction zone (Figs 3.7A and B).
- *The volume of air in this zone is approximately **150 ml** and is known as the **anatomical dead space.***

Bronchial Tree

- The right main bronchus leaves the trachea at an angle of 25° from the vertical. It enters right lung opposite to 5th thoracic vertebra (T5). *The right upper lobe bronchus emerges just* ***2.5 cm*** *from the carina.*
- This position promotes both a greater incidence of aspiration of foreign material into the right lung and accidental right main bronchus intubation when an endotracheal tube is advanced too far into the trachea (Fig. 3.8).
- One more clinically important anatomical factor is the difference in the size of lumen of trachea at the proximal end (just at glottis and sub-glottic level). In adult trachea, the narrowest portion is the glottic opening where as in children; the narrowest portion is the cricoid ring (Fig. 3.8).

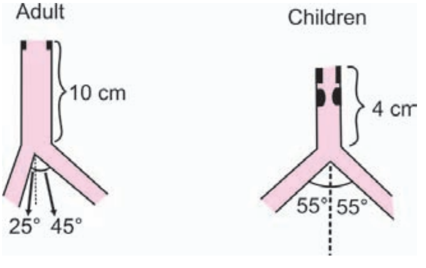


Fig. 3.8: The difference in the angle of bifurcation of trachea between an adult and a child

- ❖ More possibility of right main bronchus intubation as it is in line with trachea.
- ❖ In an adult, the narrowest portion of the airway is the laryngeal inlet.
- ❖ In a child, the narrowest portion of the airway is at the level of cricoid ring.

- Hence in children, an endotracheal tube that passes easily through the glottic opening may not pass through the cricoid ring. The practical, clinical significance of this fact is an endotracheal tube which passes easily through the larynx may not pass through the cricoid ring; if too big a tube is passed forcefully it will result in oedema and stridor after extubation.
- **The left main bronchus** is longer than the right and narrower and lies more horizontal to the trachea. *The length before the origin of upper lobe bronchus is **5 cm**.*
- It leaves the trachea at an angle of about 45° and it enters the lung opposite T6.
- In children *under the age of three years*, the angle of bifurcation of trachea into right and left main bronchi at carina is equal on both sides. Usually at an *equal angle of 55°* (Fig. 3.8).
- The point where the bronchi, nerves, lymphatic vessels, and blood supply leave the mediastinum and enter the lung is known as *the hilum.*
- After penetrating the lung, the **right main bronchus** divides into ***three lobar bronchi*** that lead to ***upper, middle, and lower right lung lobes***. They further divide into segmental bronchi (Total 10 segments).
- Right upper lobe has three segments: ***Apical, posterior, and anterior segments.***
- Right middle lobe has two segments: ***Lateral and medial segments.***
- Right lower lobe has five segments: ***Apical, medial basal (cardiac), anterior*** basal, ***lateral*** basal and ***posterior*** basal segments.
- The **left main bronchus** divides into two, ***upper and lower lobe bronchi***. There is a ***lingular lobe bronchus*** also. They also divide into segmental bronchi (Total 9 segments).

- Left upper lobe has five segments:
  - Upper division bronchus: ***Apical, posterior,*** and ***anterior*** segments.
  - Lower division (Lingula): ***Superior*** and ***inferior segments.***
- Left lower lobe has three segments: ***Apical, anterior*** *basal*, ***lateral*** *basal* and ***posterior*** *basal* segments.

Medial basal is absent as its place is occupied by the heart. Now there is a controversy among anatomists that there is a medial basal segment also.

- Lobar bronchi bifurcate and trifurcate into segmental bronchioles or terminal bronchiole that lead to lung segments.

The bronchioles lack cartilage and are made of connective tissue that contain elastic fibers and limited smooth muscles. This layer is called as 'myoelastic layer' as there are interlacing elastic fibers. The smooth muscle fibers are arranged in such a peculiar pattern, that when they contract, that not only narrows the lumen, but shortens the bronchiole also. Therefore, when they contract the length as well as the diameter gets reduced (Fig. 3.9). This is known as "***Geodesic Pattern"*** of the smooth muscles in the bronchioles.

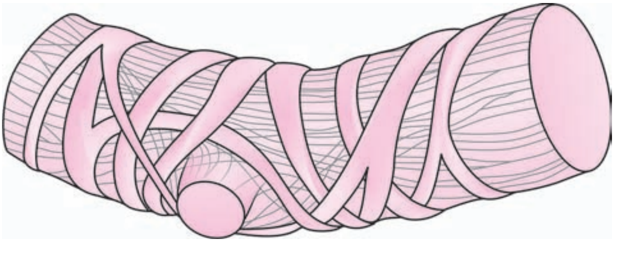


Fig. 3.9: The elastic fibers and smooth muscles arranged in a special pattern

The bronchioles are held open by the radial traction from the elastic recoil forces of lung tissue. With lack of supporting cartilage, these airways are susceptible for narrowing by bronchospasm.

Respiratory Zone (Alveolate Region)

This zone starts from the 17th generation (division) of the bronchial tree (Fig. 3.7A) and is made up of the following structures.

- Terminal bronchiole
- Respiratory bronchiole
- Alveolar ducts or alveolar passages
- Atria
- Air sacs
- Air cells
- ***Alveolar buds*** begin to appear on the walls of the transitional airways, or ***respiratory bronchiole,*** which make up the seventeenth through nineteenth generation of airway branches.
- Cartilage is absent in bronchi less than 1 mm diameter. *Elastic tissue* is distributed throughout the lung parenchyma and *is responsible for passive expiration.*
- The *terminal respiratory unit*, or *acinus* or *primary lobule*, is that portion of the lung arising from a single terminal bronchiole.
- ***The acinus*** is the primary gas-exchanging unit of the lung, consisting of the ***respiratory bronchiole, alveolar ducts, atria, air sacs, and the alveolar cells*** (Fig. 3.10).
- *The respiratory bronchiole is devoid of cilia, but above this level they are in plenty.* Each cell has about 200 cilia that have wave like motion resembling the cornfields during breeze, beating upwards towards the mouth.
- This transports the foreign material towards the upper airway. The mucociliary transport is depressed by

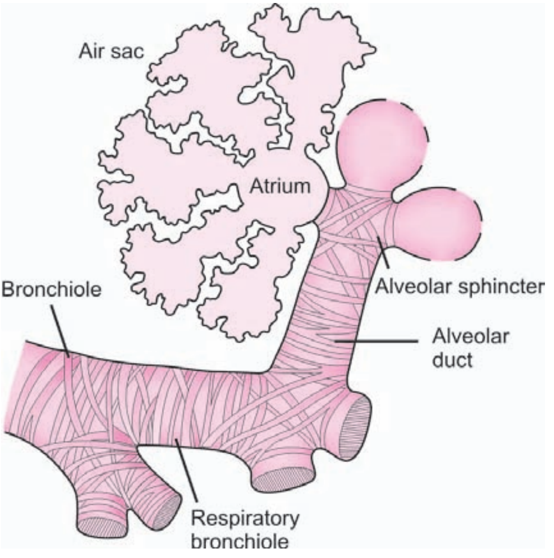


Fig. 3.10: The acinus—Divisions beyond terminal bronchiole up to alveoli

anesthesia, dehydration and drying due to lack of *humidification.*

- Through the number of airway divisions, or generations from trachea to alveolar sac is generally 23 (Figs 3.7A and B) the distance from the terminal bronchiole (Divisions 16) to the alveolus is only 5 mm, *but the respiratory zone makes up most of the lung, its volume being about 3000 ml*.
- The average diameter of an alveolus is about 0.2 mm at functional residual capacity (at resting level), though the size obviously vary with the state of inflation of the lung.

- There are approximately 300 millions alveolar capillary units (Fig. 3.11) in the adult lung. The total surface area the lung parenchyma is 50 to 100 $m^{2,}$ about the size of a tennis court.

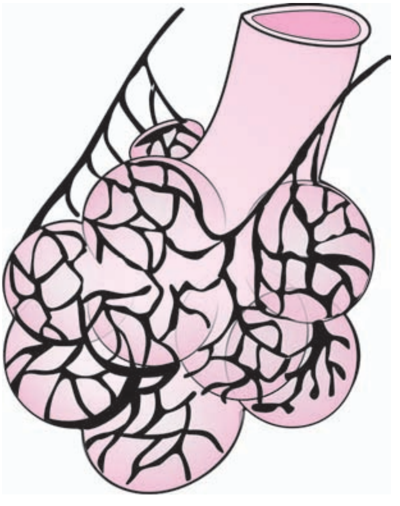


Fig. 3.11: Alveolar unit surrounded by pulmonary capillaries

- About *1500 miles of capillaries* distribute blood to these *300 millions of alveoli* in an adult.
- The blood and air is separated by the four layered alveolar capillary membrane which is less than 2 microns thick. The layers are 1. Alveolar lining fluid, 2. Alveolar epithelium, 3. Interstitial layer (this accounts for the about half the thickness of the blood-gas barrier.), 4. Capillary endothelium.
- After this, the gas has to cross a fifth layer of a thin layer of plasma to reach the RBCs (Fig. 3.12).

- The distance between the alveolus and capillary is less than the diameter of a single red blood cell (Fig. 3.12). The alveoli are surrounded by capillaries so dense that, when fully recruited they form a complete sheet of blood (Fig. 3.11). Small holes, known as pores of Kohn are present in the walls of the alveoli for even distribution of gas among the alveoli of an alveolar sac. This is known as *collateral respiration.*

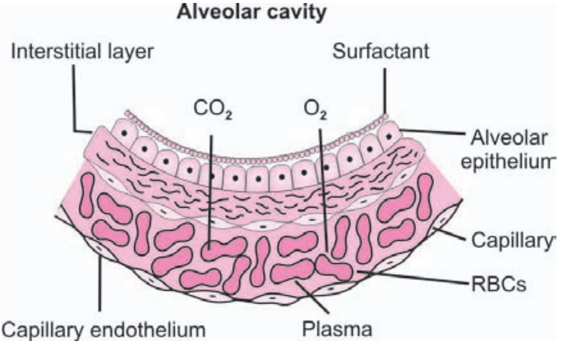


Fig. 3.12: Alveolar capillary membrane consisting of four layers

- The structure of alveolus makes it an excellent area for diffusion of gases. It has two types of cells, ***Type I** alveolar cells are squamous epithelium*, one cell layer thick that are structured to promote gas exchange and prevent fluid transudation into the alveolus. They are particularly sensitive to oxygen and inhaled agents.
- ***Type II** cells differentiate into **Type I cells*** as needed and produce surfactant, a lipoprotein that reduces the surface tension within the alveolus.

- The alveolar macrophages, free moving scavenger cells, phagocytise foreign materials that have evaded the cough reflex and mucociliary clearance system.

Surfactant

- The alveoli are lined with a monomolecular layer of the phospholipids phosphatidyl choline, which is the major constituent of surfactant. 11% of surfactant is protein. As discussed earlier, *surfactant reduces the surface tension*, helps to *stabilize the alveoli open*, and may keep the alveoli dry.
- The surfactant prevents the alveoli and bronchioles from collapsing, especially during expiration, by reducing surface tension. Surface tension is due to the fluid lining the alveoli. This fluid develops a cohesive force that tends to collapse the alveoli. Surfactant reduces the cohesive force.
- The lung therefore consists of hundreds of millions of relatively unstable bubbles, each 0.3 mm in diameter. Surfactant makes it easier to expand the lung (increases the compliance), thereby reduces the work associated with breathing. Surfactant and alveolar stability may be lost in some disease states (e.g ARDS). This loss leads to *atelectasis*, *impaired gas exchange*, and *increased work of breathing* (WOB).
- Gas exchange occurs remarkably efficient at the alveolar capillary membrane. The blood passes through the capillaries in approximately 0.5 to 0.75 seconds at rest. However, it is estimated that gas exchange is completed when the blood has traversed only one fourth of the capillary distance. This efficiency provides reserve time for gas exchange during disease and exercise states. Diffusion distance and time may be increased in alveolar congestion, interstitial or alveolar edema or pulmonary fibrosis.

Gas exchange at the alveolar-capillary membrane is known as ***external respiration,*** whereas the exchange of oxygen and carbon dioxide between the systemic capillaries and cells of the various organs systems is known as ***internal respiration***.

MECHANICS OF THE LUNG (WITH RELATION TO SURFACE TENSION)

When the lungs are removed from the thoracic cage, they tend to collapse, assuming a volume which is much smaller than their usual resting volume or functional residual capacity (FRC). This tendency is due to:

1. Elastic tissue within the lung parenchyma.
2. Surface tension at the liquid gas interface within the alveoli.

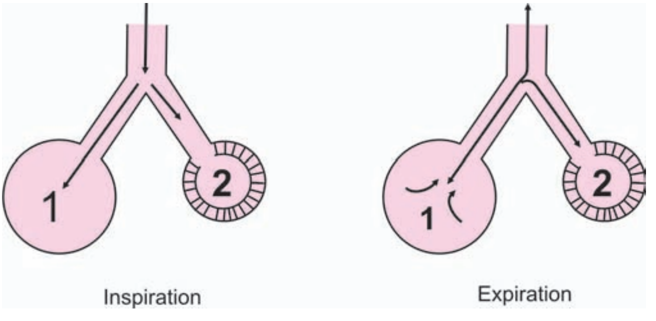


Fig. 3.13: Effect of uneven time constant on distribution of ventilation during inspiration

- ❖ Note that during inspiration, alveoli with normal time constant **1** inflate faster, whereas that with longer time constant **2** inflates very slowly.
- ❖ During expiration, the unstable alveoli with longer time constant still get inflated and part of the expired air from the normal alveoli enter into them.

If the surface tension is constant, the laplace relation dictates the pressure is highest in the smallest bubbles; if it is in continuity with a larger bubble, the smaller bubble will empty completely (Fig. 3.13).

Laplace relation (P = 2 T/R)

(P is Pressure, T is Surface Tension, and R is Radius of the bubble)

It simply means that a *smaller alveolus (unstable one)* needs higher pressure to inflate it than the neighbouring *larger alveolus (normal one).*This is explained by the experiment of blowing two balloons connected to a "Y" piece (Fig. 3.14).

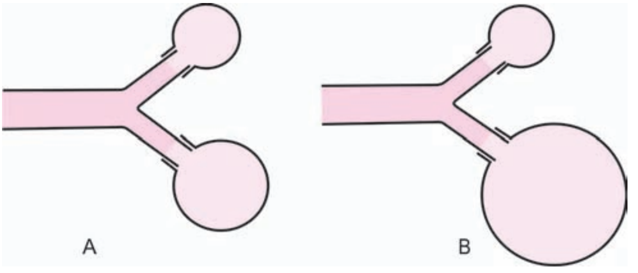
This phenomenon has to be born in mind when dealing with the ventilation of patients with *unstable lung units* as in ARDS.

In healthy lungs, the tendency of the alveoli to collapse is reduced by the presence of surfactant, as the *surface tension (T) of surfactant* decreases with decreasing alveolar size, the tendency of small alveoli to empty into larger ones is abated, and alveolar stability is ensured.

It is well known that if a person blows down the end of a Y-piece connection to which are attached two balloons, then one balloon inflates preferentially (Figs 3.14A and B). If further air is blown down the connection, one might reasonably expect that the less inflated balloon would expand. That is not the case. The already expanded balloon would expand even further.

In the case of alveoli, the unstable one has less compliance (a longer time constant) and the normal one has normal compliance (normal time constant). It will be discussed in detail in Physiology in Chapter 4.

This phenomenon is due to the law of laplace. However, if this law is applied strictly in case of lung, then theoretically a small segment of collapsed lung could never be re-expanded, as any further pressure would only continue to expand the already distended alveoli.



Figs 3.14A and B: Diagram to illustrate the law of laplace

- ❖ Same pressure is exerted to the balloons by blowing through the tube.
- ❖ Shows that one balloon gets preferentially inflated (normal); the other is less inflated (unstable).
- ❖ Shows that further inflation will inflate the normal one than the unstable one.

Experience shows that due to other physiological and anatomical factors, *this law cannot be applied rigidly.* There is no doubt that alveoli which already contain air are easier to expand than those that are completely collapsed. However, prolonged positive pressure will reinflate all portions of lung if surfactant is not deficient in the alveoli.

The remarkable property of surfactant is that, its surface tension varies directly with the amount of surface area exposed. Thus, as *the alveolus contracts*, the *tension of the lining secretion gets automatically decreased*. If it were not for this unusual property, an alveolus, once collapsed, would require greater force to re-expand it and persistent collapse would result.

Both in neonatal and adult respiratory distress syndromes *surfactant is depleted*, and the *alveoli become unstable with an increasing tendency to collapse*. Different ventilatory strategy may be employed to keep the unstable alveoli from collapsing.

NERVE SUPPLY TO THE RESPIRATORY TRACT

- The respiratory tract is innervated by autonomic nervous system, both sympathetic as well as parasympathetic.
- Parasympathetic is from vagus nerve.
- Sympathetic is from the sympathetic ganglia of T2 to T4 or T5 and also from inferior and middle cervical ganglia.
- Each vagus passes to the back of the hilum and is joined by the branches of sympathetic and forms ***the anterior*** and ***posterior pulmonary plexuses*** (Hilar plexuses). From there, fibers go to the main bronchi and pulmonary artery and their branches.
- Vagal stimulation causes bronchoconstriction and sympathetic stimulation causes bronchodilatation.
- The laryngeal irritation may stimulate the vagus and cause bronchoconstriction of mainly larger airways.
- The sympathetic receptors are mainly Beta 2 type. These receptors, when stimulated produce broncho-dilatation.

BLOOD SUPPLY TO THE RESPIRATORY TRACT

The lungs get two different blood supplies.

The Bronchial Circulation

- The bronchial arteries (one for the right and two for the left lung) is a division of systemic circulation; the bronchial arteries arise from the aorta, traverse along the bronchi, and supply the bronchi and bronchioles as far as the end of terminal bronchioles. This circulation drains into the pulmonary veins.

- This circulation receives only about 2% of cardiac output.
- *Distal to the terminal bronchioles, the blood supply is from the pulmonary artery.*

The Pulmonary Circulation

- This is actually the output from right heart. It starts from the main pulmonary artery, which receives unoxygenated blood from the right side of the heart.
- The main pulmonary artery divides into the right and left pulmonary arteries at the point where the main stem bronchi divide in the hilum.
- The arteries then divide, running parallel to each division of the airways until they finally terminate in the capillary network around the alveoli. The network is so dense that when they are fully recruited, the alveoli are nearly covered with a sheet of blood.
- The walls of the pulmonary arteries are very thin. They contain smooth muscles, but are less strong than that in the arteries of systemic circulation and carry deoxygenated blood. Therefore, they are very compliant to accommodate a large volume of blood in case of need.
- The oxygenated blood from these arteries is collected by the pulmonary veins, which unite with other veins eventually forming the four pulmonary veins that drain into the left atrium.

The Lymphatic Drainage

- This lymphatic system consists of lymph channels and nodes. The channels are present in the pleura and in the peribronchial and perivascular spaces forming a network around the blood vessels that they accompany.

- The lymph is the fluid that is formed by the normal process of interstitial fluid development.
- It flows from the periphery towards the main lymphatic channels along the bronchial tree towards the lymph nodes clustered around each hilum, from there towards either the thoracic duct or right lymphatic ducts which drain into the right and left subclavian veins.
- The channels contain valves that promote unidirectional flow.
- The lymph nodes are scattered throughout the course of the lymphatic channels.
- These nodes are the lymph filtering stations; large particulate matter may be deposited in them.

The lymphatic system has two main functions:

- Maintaining **fluid homeostasis** of lungs and pleural cavity.
- Providing **immunological defence** mechanism.

Fluid Homeostasis

- It reabsorbs the excess fluid in the interstitial layers of lung, peribronchial spaces, and pleural spaces and thus returns the serum, escaped plasma proteins, and products of cellular metabolism that cannot be absorbed by the capillaries to the circulation.
- By reducing the interstitial protein concentration, the lymphatic system decreases interstitial colloid oncotic pressure and assists in prevention of pulmonary edema formation.

Immunological Defence

- It filters out bacteria and other harmful substances that have escaped the mucociliary escalator.

- Filtering done at the lymph nodes before the lymphatic fluid is returned to the general circulation, thereby protecting the body from dissemination of foreign material.

LUNGS

Lobes and Segments

- The lungs are somewhat in conical shape. The apices are behind the inner third of clavicle raising 2–4 cm above it. The bases rest on the superior surfaces of the diaphragm.
- The right lung, which accounts for about 55% of the lung function, has three lobes: *Upper, middle and lower.*
- The left lung has two lobes**:** *Upper and lower*. However, the left upper lobe has a superior and inferior division. The inferior portion is known as ***lingula*** and is thought as being comparable to the middle lobe.
- The lobes of the lungs are further divided into broncho-pulmonary segments. Each lung has ten segments numbered universally from number one to ten. Each segment has ***its own airway, arterial supply, and venous drainage***, which allows any diseased segments to be surgically removed.
- Understanding the anatomy of various broncho-pulmonary segments is essential to apply it for pulmonary toilet technique of postural drainage, percussion and for anatomically defining a lesion involving the segments.

Pleura and Pleural Space

- Each lung is invaginated from the hilum into the closed sac of pleura. So of the two layers of the sac, the outer

one is attached to the inner surface of thoracic cage and the inner layer is attached to the surface of lungs. These layers are known as pleural layers.

- The lungs and thoracic cage are lined with these layers of a continuous sheet of elastic collagenous fibers that is described as; the ***visceral pleura*** and the ***parietal pleura*** respectively.
- The visceral pleura is a thin lining around the lungs, the lung fissures and hilar bronchi and vessels.
- The parietal pleura lines the inner surface of the thoracic cavity. *The parietal pleura has nerve receptors for pain,* where as the visceral pleura does not have it.
- A mucous solution is produced by the cells of pleura. This solution, which is probably **less than 10 ml**, lubricates the pleural surfaces, allowing smooth movement of the surfaces one over the other. It also holds the two surfaces of pleura together by means of surface tension. Thus the pleura move in unison with the thoracic cage during inspiration.
- Surface tension is the attractive force between adjacent liquid molecules and this force preserves the integrity of the surfaces, preventing it from separating.
- *It is this surface tension between the two layers of the pleurae, opposing the tendency of the lungs to collapse because of its elastic recoil, which leads to the existence of a negative pressure of about **–5 mm Hg** within the intrapleural space.*
- The pleural space is essentially a potential space between the two layers of the pleura. If excess fluid collects in the space (pleural effusion) or air enters the space (pneumothorax) the lung expansion is hampered.
- If the lung expansion is significantly compromised by such collections, a chest tube may have to be introduced to drain the fluid or air under a water seal.

- If an injury to the chest wall has caused an opening in the thoracic cage, the air may enter the intrapleural space and the lung, because of its elastic recoil collapses.
- *The pleural cavity in left and right side are entirely separate* and the disease affecting one pleural cavity will be restricted to that hemithorax.

Thoracic Cage

- The lungs are housed in the thoracic cage, which is bordered posteriorly by the vertebral column, anteriorly by the sternum and laterally by the ribs.
- The floor is formed by the dome shaped diaphragm. The ribs, 24 in number, 12 on each side, are attached posteriorly to the spinal column and then extend around and down, under the arms, where they turn upward again and extend not as bone but as cartilage to reach the sternum. These resilient bars of cartilage are known *as costal cartilages.*
- The first seven pairs of costal cartilages directly reach the sternal border directly to form sternocostal junctions whereas, that of the eighth, ninth and the tenth pair of ribs unite together running obliquely to join the cartilage of the seventh rib.
- The last two pair of ribs are known as floating ribs, they don't have attachment anteriorly.
- The ribs provide not only protection for the lungs, but also give attachment for respiratory muscles.
- The sternum has three parts: The manubrium sterni above, the body of the sternum in the middle and the xiphoid process below. The point where the manubrium and the body articulate, the sternal angle, is a prominent ridge that can be felt under the skin. At this point the

second rib articulated with the sternum and is a land mark for counting the ribs and the intercostals spaces.

- The intercostals spaces are numbered according to the number of rib above, so the *space above the second rib is the first intercostals space.*

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CHAPTER 4

Applied Physiology of Respiration

- *External respiration*
- *Internal respiration*
- *Oxygen transport and oxygen dissociation curve*
- *Control of respiration*
- *Ventilatory response to carbon dioxide*
- *Ventilatory response to hypoxia*
- *Protective mechanisms in respiratory system*
- *Symbols used in respiratory physiology and mechanical ventilation*

The foundation of the care for the patient supported by mechanical ventilation begins with the understanding how spontaneous ventilation is achieved. The knowledge of normal respiratory physiology must be used as a tool to interpret the changes that occur in pathological states. This will be of help to choose the appropriate mode of ventilator therapy suited to various patients with different pathological conditions. It may also help us to evaluate the benefits of the therapy.

Physiological facts about respiration:

- The basic purpose of breathing is to take up oxygen from the atmospheric air in the lungs and from lungs into the blood and to feed all the tissues for burning the food to generate energy for life.
- The second purpose is removing the CO_2 from the lungs, a byproduct of metabolism that has reached the lungs through the blood.
- In normal healthy individuals, the respiration occurs about 12 to 14 times per minute.
- It has two phases; one is inspiration when air enters the lung and the other phase is expiration where the air is sent out of lungs.

- After expiration there is a short pause before the next inspiration is started. This is *expiratory pause* and it is included in the expiratory time.
- Normally inspiration is short and expiration is longer, the time ratio is usually 1:2.
- Inspiration is done by active contractions of the respiratory muscles which create a negative pressure in the alveoli that is open to the atmosphere through the tracheo bronchial tree. The atmospheric air flows into the lungs.
- Expiration is passive, which means when the respiratory muscles stop contracting, the elastic recoil of the lungs and thoracic cage compresses the alveoli to squeeze and expel the gases out.
- The most important aspect of respiration is that in normal conditions of life it happens by the control of the respiratory center, modulated by various reflexes related to the regulation of respiration. *It is so effortless that obviously no one normally realizes that breathing is going on.* Only when there is a problem, respiration is realized as a work and that increased work of breathing is very unpleasant and distressing. It is abnormal (pathological). However, unlike other vital functions, respiration could be controlled to some extent by voluntary efforts.
- The other vital functions, particularly the cardiovascular function is intimately interlinked with respiration and so any interference in this function either by disease or by artificial ventilation is likely to have adverse effect on the cardiovascular system also.

COMPONENTS OF NORMAL RESPIRATION

- Respiration is simply the gaseous exchange between an organism and its environment. Oxygen is absorbed and carbon dioxide is excreted which involves two components.

- ***External respiration*** is moving the air in and out of lungs and the gaseous exchange *between air and blood interface* at the level of alveolar capillary membrane.
- ***Internal respiration*** is the utilization of oxygen for the metabolic processes and the release of carbon dioxide as a metabolite at tissue level.
- Nevertheless, physiologically diffusion of gases at alveolar capillary membrane is referred to as ***external respiration*** and diffusion of gases across the cell membrane is called as ***internal respiration*** (Fig. 4.1).
- This carbon dioxide from the tissues is carried by the circulation to the lungs to be eliminated by external respiration. For adequate gaseous exchange, the lung has to be perfused well with deoxygenated blood from pulmonary artery.
- Hence, there are the three essential integral parts of ***external respiration*** namely;
  - ***Ventilation*** (moving air in and out of the lungs)
  - ***Perfusion*** (adequate pulmonary flow into all parts of the lung)
  - ***Diffusion*** (exchange of gases across the alveolar capillary membrane).
- By artificial ventilation, the external respiration can be modified to a greater degree. Here again modification of perfusion has its limitations.
- *However, mechanical ventilation cannot modify the internal respiration at the tissue level, if it is deranged.*

SOME PHYSIOLOGICAL FACTS OF GAS EXCHANGE

Diffusion

The first step of external respiration is air reaching the alveoli. Once it is accomplished, the next essential process is diffusion starts.

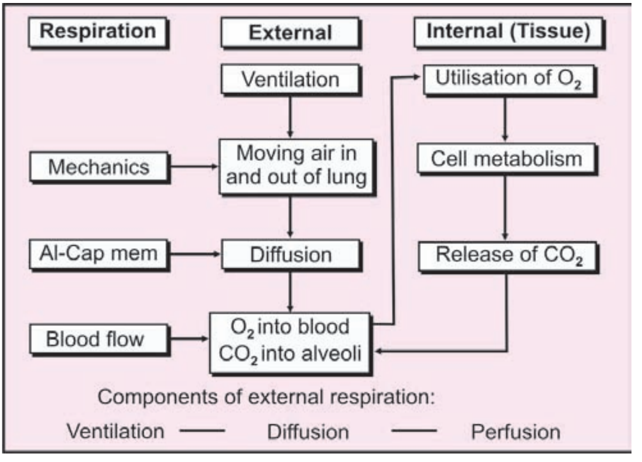


Fig. 4.1: Internal and external respiration with their integral parts

- Diffusion is the movement of gas molecules from an area of higher partial pressure to an area of lower partial pressure.
- Here in the alveoli, the driving force for diffusion is *the pressure gradient across the alveolar capillary membrane.*
- The microscopic anatomy of the alveolar capillary membrane and the pathway for the gas molecules to reach the RBCs and back has been described in Chapter 3 (Fig. 3.12).
- The factors that could modify the diffusion across the alveolar capillary membrane are;
  - The surface area of the membrane
  - The thickness of the membrane
  - The pressure difference (gradient) between the two sides of the membrane
  - The diffusion coefficient of the gas (how readily the gas diffuses in relation to the other gases in solution).

- The lung has tremendous surface area for gas exchange, approximately 70 square meters. It has been shown that a total area of five healthy segments out of the available 19 segments is enough for gas exchange at rest. However, extensive parenchymal diseases may reduce the surface area of A-C membrane.
- The thickness of the A-C membrane may be increased causing difficulty in diffusion may occur in; chronic fibrotic lung diseases, pulmonary oedema etc.
- Though both oxygen and carbon dioxide can diffuse readily diffuse across the A-C membrane, carbon dioxide is highly diffusible than oxygen about 20 times more diffusible. Hence, the factors that affect the diffusion of oxygen may not affect the diffusion of carbon dioxide.
- Gas exchange occurs remarkably efficient at the alveolar capillary membrane. The blood passes through the capillaries in approximately in 0.5 to 0.75 seconds at rest. However, it is estimated that gas exchange is completed when the blood has traversed only one fourth of the capillary distance. This efficiency provides reserve time for gas exchange during disease and exercise states (Fig. 4.2). Diffusion distance and time may be increased in *alveolar congestion, interstitial or alveolar oedema, or pulmonary fibrosis.*
- One of the great advantages in the process of diffusion across A-C membrane is this enormous reserve time available, that allows adequate diffusion even in hyper dynamic states like severe exercise, pyrexia, high cardiac output diseases, etc. and only in severe damage due to diseases, diffusion is compromised.
- The other factor is the *pressure gradient across the membrane*. A quick review of the percentage and the partial pressure of various gases in the atmospheric air, expired air and alveolar air will help to discuss about it.

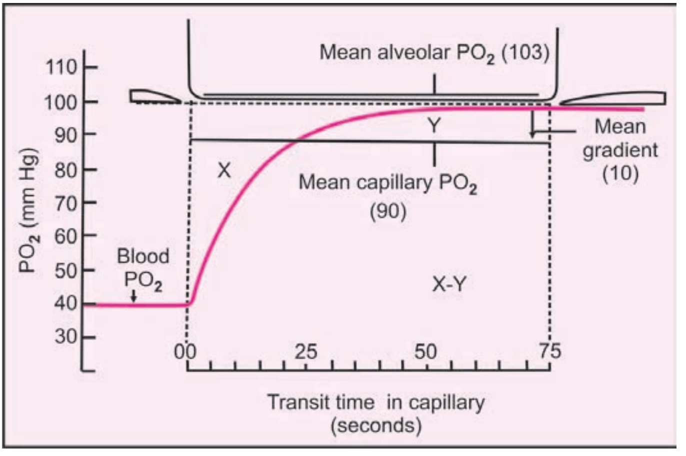


Fig. 4.2: Transit time of blood in pulmonary capillary

- The time taken for the transit is 0.75 seconds.
- Partial pressure (PO_2) from 40 mm Hg to 97 is reached in 0.25 seconds.
- Full saturation would occur in this time.
- The remaining 0.5 seconds is the reserve.

The percentage of oxygen, carbon dioxide, and nitrogen in air and respiratory tract:

	Atmospheric air	*Alveolar air*	*Expired air*
Oxygen:	20.94%	14.2%	16.3%
CO_2	0.04%	5.5%	4.0%
Nitrogen:	79.02%	80.3%	79.7%

The approximate average values of partial pressures of oxygen, carbon dioxide, and nitrogen in atmospheric air, expires air, alveolar air, arterial blood and venous blood (Fig. 4.3).

	Atmospheric air	*Expired air*	*Alveolar air*	*Arterial blood*	*Venous blood*
Oxygen	159.0	150.0	100	96	40
CO_2	0.3	0.3	40	40	46
Nitrogen	600	563	573		
H_2O	5	47	47		

- Carefully looking at it, it is found that the difference in partial pressures of oxygen and carbon dioxide in alveolus, arterial blood, and venous blood could be marked.
- Gas exchange has to occur between alveolar air on one side of A-C membrane and blood traversing through pulmonary capillary on the other side.
- Partial pressure of oxygen in alveolus is 100 mm Hg and that in pulmonary capillary is 40 mm Hg. Thus *a pressure gradient of 60 mm Hg* is available for oxygen to diffuse into the pulmonary blood to reach equilibrium.
- Similarly *a pressure gradient of 6 mm Hg* (46 mm in venous blood and 40 mm in alveolus) allows easy diffusion of carbon dioxide as it is 20 times more diffusible than oxygen into the alveoli.

Alveolar PCO_2 (P_ACO_2)

- Oxygen uptake at rest is normally 250 ml/minute and carbon dioxide production is 200 ml/minute.
- CO_2 diffuses from pulmonary arterial blood to alveolar gas, where the alveolar partial pressure of CO_2 (P_ACO_2) is determined by its rate of transfer and its dilution by *alveolar ventilation*.
- In other words alveolar ventilation determines the alveolar carbon dioxide and so doubling the alveolar ventilation will halve the alveolar PCO_2. Halving

ventilation will double the PCO_2, although equilibrium in this case is reached slowly (Up to 15 minutes) because of the limited metabolic production of CO_2.

- During apnea, $PaCO_2$ rises at a rate of 3 to 6 mm Hg/ minute.

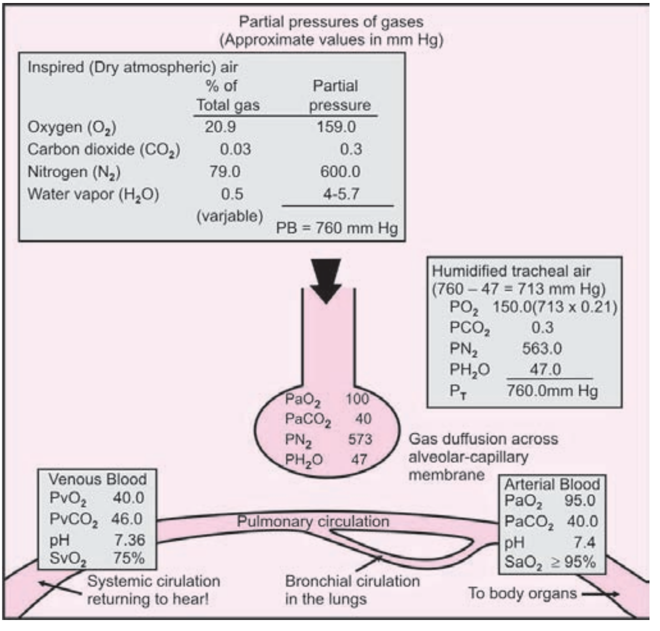


Fig. 4.3: Partial pressures of various gases in atmosphere, alveolar air, and blood

Alveolar PO_2 (P_AO_2)

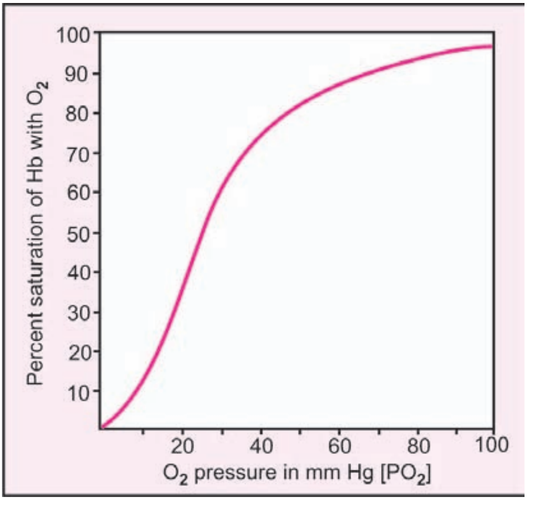
- The main factor which influences this is the barometric pressure. (PB) Normal is 760 mm Hg.
- Normal saturated water vapour in the alveoli at body temperature is 47 mm Hg. This is constant and has to be subtracted from the PIO_2. The barometric pressure

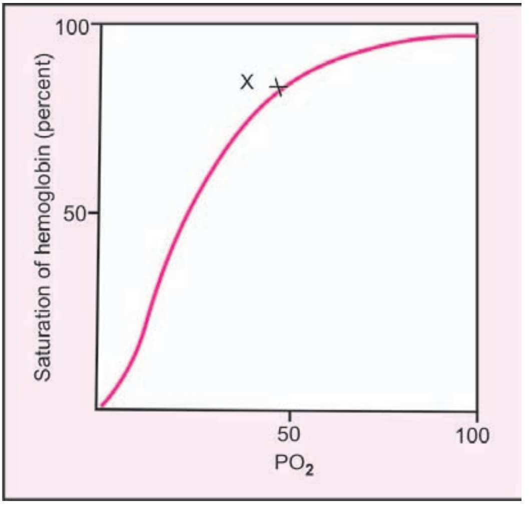
decreases as we go to high altitude, the barometric pressure continues to drop and at 63,000 ft the barometric pressure is 47 mm Hg and so PIO_2 becomes zero, the blood boils.

- Next factor is cardiac output. If cardiac output is low, the perfusion to lung is less, the uptake of oxygen is less, and so P_AO_2 will increase.
- In shivering patient oxygen consumption will be doubled, so uptake will be more and P_AO_2 will be low.

Oxygen Carriage in Blood

- Barcroft and Poulton described oxygen dissociation curve of hemoglobin. They showed how at 37 °C, and with PCO_2 of 40 mm Hg and pH of 7.4 the saturation of hemoglobin changes with PO_2.
- The ***saturation of oxygen*** in hemoglobin for varying partial pressures of oxygen to which it is exposed is ***not linear.***
- It showed that at normal partial pressure value of 90 to 100 mm Hg, hemoglobin is 97% saturated with oxygen, at 60 mm Hg saturation is still 90%.
- Venous blood has 40 mm Hg and saturation is 75%.
- **The** *oxygen dissociation curve* ***compares the partial pressure of oxygen with the percentage saturation of hemoglobin*** **(Fig. 4.4.A).**
- *It is important to note that because of the special 'S' shape of the curve, it is possible for the PO_2 to be reduced with minimal effect upon the saturation*. However when the point X is reached, the saturation falls rapidly with only a small decrease in PO_2 (Fig. 4.4 B).
- Upon close consideration of this curve, it will become apparent that shift of the curve to the left or right will have a pronounced effect upon the availability of oxygen to the tissues.





Figs 4.4A and B: Oxygen dissociation curve

- *This dissociation curve provides a safety of allowing a considerable fall in partial pressure of oxygen in blood, but maintaining a high saturation, which means the transport of oxygen to the tissues, is still done safely.* ***Moreover, at a lower level of saturation hemoglobin readily parts with the oxygen to the tissues.*** This is the greatest advantage for oxygen delivery to tissues.
- Careful analysis of this curve makes it clear that if the curve *is shifted to left*, oxygen dissociation to the tissues is relatively less and so it is disadvantageous, whereas if it *is shifted to the right*, it easily parts with its oxygen, so it is advantageous.
- **P 50**: This is the value of oxygen tension (partial pressure) needed for 50% saturation of hemoglobin, normally is 26 mm Hg and is a way of describing any shift of the dissociation curve to the right or left.
- Such shifts occur with, acidemia (to right), alkalemia (to left), high pCO_2 (to right), higher temperature (to right), decrease in 2, 3 Diphosphoglycerate (2, 3 DPG) in red cells (to left), increase in 2, 3 DPG as in anemia, chronic hypoxemia, stored blood etc (to right).
- 1 gm of hemoglobin carries 1.34 ml of oxygen when fully saturated. To this 0.3 ml of oxygen carried in the form of physical solution may be added.
- Therefore, normal arterial blood has an oxygen content of 20 ml/dl. The venous blood has 15 ml/dl. The arteriovenous oxygen difference is about 5 ml/100 ml.
- If oxygen is breathed at 3 atmospheric pressure, this 5 ml of oxygen/100 ml of blood can be carried in the form of physical solution. Hence there is a theoretical possibility of delivering adequate oxygen to the peripheral tissues with out hemoglobin carrying oxygen.
- Consciousness is likely to be lost in a normal person with a PaO_2 below 27 mm Hg.

Cyanosis

- When the amount of reduced hemoglobin, which is darker red than oxygenated form, exceeds 5 grams per 100 ml, a bluish hue can be seen. This is termed ***cyanosis.***
- Cyanosis occurs when the capillary blood has a reduced hemoglobin level over 5 gm/dl. Best detected in lips and buccal mucosa.
- If the hemoglobin levels are normal, it can be detected at arterial saturations of 95–90% (PaO_2 50 to 60 mm Hg), although there is much variation. If the hemoglobin level is low, it may not be detected however low the PaO_2.
- Cyanosis may be produced by any one of the following types of hemoglobins; 5 gm/dl of reduced hemoglobin, 1.5 gm/dl of methaemoglobin or 0.5 gm/dl of sulphaemoglobin.
- It may not be apparent in anaemic subjects, in carbon monoxide poisoning (because carbon monoxide and hemoglobin combination – carboxyhemoglobin is bright red) and in histotoxic hypoxia.
- Cyanosis may be ***central,*** as seen in the mucous membranes of the lips or ***peripheral*** as seen in the nail beds, for example, on exposure to cold, in patients with circulatory disorders.

Oxygen Flux

This is the rate of oxygen carriage in arterial blood. In other words, it is the amount of oxygen leaving the left ventricle per minute in the arterial blood. It can be calculated by:

- Cardiac output (ml/min) × SaO_2 × Hb concentration (g/ml) × 1.34.
- Normally it is 1000 ml/min.
- Values below 400 ml/min are probably lethal, if continued for long time.

Clinical Significance

- Out of this 1000 ml normally about 250 ml of oxygen is used up in cellular metabolism and the rest returns to the lungs in the mixed venous blood, which is therefore about 75% saturated with oxygen.
- Because the three variables in this equation (cardiac output, oxygen saturation, and hemoglobin concentration) are multiplied, a relatively trivial reduction of each may result in catastrophic reduction in oxygen flux, leading on to hypoxia.

Carbon Dioxide Transport

- The proteins in plasma (in the red cell mostly as hemoglobin) combine with carbon dioxide to form carbamino compounds.
- The rest of the carbon dioxide is transported either in simple solution or as bicarbonate ion.
- In normal circumstances, 100 ml of blood carries 3 ml of carbon dioxide.
- Carbon dioxide is highly diffusible, about 20 times greater than oxygen.
- As discussed earlier, pulmonary arteriole carries deoxygenated, blood where the PCO_2 is 46 mm Hg. The alveolar CO_2 (P_ACO_2) is 40 mm Hg. Because of high diffusibility, gradient of 6 mm Hg causes efficient diffusion into the alveoli.
- However, for the elimination of carbon dioxide alveolar ventilation is the absolute requirement.

Central Response to CO_2

- Inhalation of 5% carbon dioxide, though unpleasant, can be inhaled for long periods without ill-effects.

- However, unconsciousness inevitably supervenes when concentration is raised to 15%. At this level, muscle rigidity and tremors may be observed.
- At concentration of 20–30% generalized convulsions can be produced.

CONTROL OF RESPIRATION

Respiration is controlled through interplay of many complex processes to ensure that adequate oxygen is available for metabolism and that the byproduct of cellular metabolism, carbon dioxide is removed and acid-base homeostasis is maintained. It is mainly by humoral control through the action of chemical stimuli on the regulatory centers in the brainstem.

Voluntary Control by Cortical Centers

- Normally breathing continues rhythmically without our conscious effort in such a way that no one ever realises that he is breathing. This rhythmicity of respiration without conscious effort occurs during sleep, or during concentration of work, etc. but conscious voluntary control of breathing is possible.
- Conscious regulation of breathing is mediated by the cerebral cortex, specifically in the motor cortex and the limbic area.
- An individual may consciously hyperventilate, hold his breath, or alter the pattern of breath for sniffing, singing, speaking, humming or to perform isometric work such as straining at stool and lifting a weight. However, the ability of the cerebral cortex to override on the respiratory centers is limited. For example, holding one's breath to the point of unconsciousness is limited by carbon dioxide stimulation of spontaneous breathing.

- Many higher centers exert some influence on respiration; for example, the acts of crying, laughing, swallowing, speaking, and coughing require a careful integration of the mechanical systems. The impulses arising from the cortical and thalamic areas influence the changes in respiratory pattern.

The Respiratory Centers

- Respiration is a cyclical event brought about by rhythmical discharges from the respiratory center.
- The cortex is responsible for voluntary control, the pons and medulla for automatic control.
- The rhythm of breathing is controlled by the respiratory center in the floor of the fourth ventricle. These are not well defined centers, but groups of neurons controlling specific activity of respiration.
- There are three main parts:
  1. ***Inspiratory Center, a*** *medullary center* which can initiate and maintain the sequence of breathing.
  2. ***Apneustic center,*** in the middle and lower pons, which if unopposed tends to produce inspiratory spasm or apneustic (continuous inspiration) breathing.
  3. ***Pneumotaxic center,*** in the upper third of pons that restrains the apneustic center periodically.
- In addition to these, there is one ***Expiratory center,*** situated in the ventral side of medulla on either side, when stimulated, produce expiratory muscle activity.
- Expiration is normally a passive process during quiet breathing but becomes active during exercise.
- The medullary center is the main coordinating center for all sources of input from other centers involved in voluntary control.

- *The pneumotaxic center encourages rhythmic ventilation by limiting inspiration.*
- There is much uncertainty concerning the origin of respiratory rhythmicity. It is generally thought that there is an inherent rhythmicity in groups of neurons in the brainstem which is modified by their afferent inputs.
- Even this idea is questioned and it may be that there is no inherent rhythmicity, the respiratory center is merely transducing the afferent input into rhythmic output, but in the absence of sufficient afferent input remaining silent.
- *The main input is in the form of stimuli from various areas caused by increased CO_2.*
- Input to these centers come from proprioceptors and peripheral chemoreceptors via the glossopharyngeal and vagus nerves.
- Output (efferent) activity is through phrenic nerve to diaphragm and thoracic nerves 2 to 12 to the intercostal muscles.

Chemical Control of Breathing

- *Changes in arterial PCO_2* will either stimulate or depress the center. The center normally maintains the PCO_2 at 40 mm Hg. If it falls to 30–34 mm Hg, respiratory drive usually ceases, leading to apnea.
- PCO_2 has much more effect upon the respiratory center than PO_2 and in fact, this is the normal stimulus to respiration (via the pH of CSF).
- This system is extremely sensitive because carbon dioxide is a very soluble gas and crosses the biological membranes easily.
- On entering the CSF it forms carbonic acid (H_2CO_3) and thereby alter the hydrogen ion concentration (and pH) of the CSF.

- The effect of $PaCO_2$ on the respiratory center is modified by the CSF bicarbonate concentration.
- The change in CSF pH is the primary stimulus. The respiratory center usually takes about 30 seconds to respond to the change in $PaCO_2$.
- The usual pathway is influenced to a certain extent by central ***chemoreceptors*** in the medulla.
- The central chemoreceptors are very sensitive to the pH changes of the cerebrospinal fluid (CSF) and blood.
- A fall in pH (rise in acidity) of the blood or a fall in the PO_2 will increase the respiratory center drive, while the reverse changes inhibit the center, but to a lesser degree. These mechanisms result in the maintenance of arterial PCO_2 within narrow limits.
- This mechanism constitutes a 'feed back' loop, thus providing 'fine tuning' of the system (Fig. 4.5).
- This feed back has a very high significance when ventilating the patient artificially whether it is during anaesthesia or for therapeutic ventilation. Therefore, it is essential that the $PaCO_2$ is maintained around 40 mm Hg.

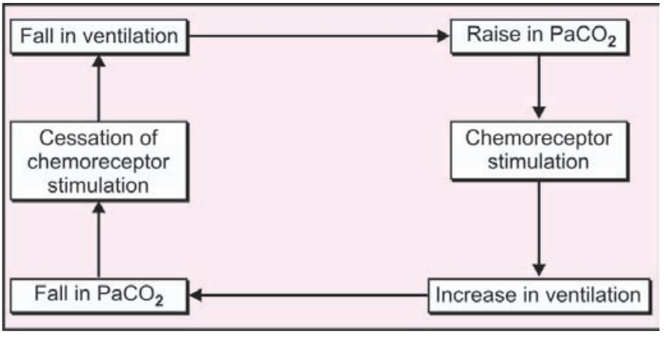


Fig. 4.5: Feed back stimulation of ventilation through chemoreceptors

Clinical Significance

- A patient on ventilator with *larger tidal volume* than needed *with or without higher rates of ventilation* will quickly develop *hypocarbia,* which will lead to difficulty in the weaning process.
- Similarly, during anesthesia, if moderate hyperventilation is maintained, it is possible to lower the $PaCO_2$ to a level near 36 mm Hg. *This low $PaCO_2$, together with the depressed respiratory center by the opioids with reduced sensitivity for the stimulation by CO_2, will* ***leave the patient apneic in spite of being awake.***
- There is a direct relationship between alveolar PCO_2 and ventilation such that if the PCO_2 is raised so is the alveolar ventilation, both by an increase in tidal volume and respiratory rate.

Central Chemoreceptors

- Bilateral superficial chemoreceptors on the ventrolateral surface of the medulla are called as *medullary H^+ chemoreceptors.*
- It is situated superficially near the surface of medulla, and is sensitive to the H^+ concentration of the interstitial fluid bathing it.
- These are influenced by the PCO_2 of the CSF and the cerebral capillary blood and by the bicarbonate concentration of the blood.
- The bicarbonate concentration in the blood has no immediate effect on the medullary H^+ chemoreceptors due to the diffusion barrier for bicarbonate ions which exists between blood and CSF and between the blood and brain.
- Carbon dioxide, being rapidly diffusible throughout blood, tissue, and CSF, provides the means whereby

rapid changes in the H^+ concentrations of the CSF and the fluid surrounding the receptor can be produced.

The Peripheral Chemoreceptors

- The peripheral chemoreceptors consist of *carotid and aortic bodies.*
- The carotid bodies are 5 mm in diameter and are situated at the bifurcation of the common carotid arteries. They are innervated by the sinus nerves which are the branches of glossopharyngeal nerves.
- **The most striking aspect of the anatomy of the carotid body is its vascularity. It has by far** the highest blood flow of any organ in the body; 2000 ml/100 gm/min.
- The aortic bodies situated close to the aortic arch of aorta, are similar but they appear to be less important as chemoreceptors but may be important for their cardiovascular effect.
- They respond to changes in PO_2, PCO_2, and the pH of the blood supplying them. Of these, the most important is a fall in arterial PO_2.
- The respiratory response to hypoxia is mediated entirely through peripheral chemoreceptors.
- These receptors *are stimulated when the arterial oxygen tension falls below 60 mm Hg*. However, the effect of hypoxia *on the respiratory center is depression.*
- Any increase in ventilation due to hypoxia tends to lower the arterial PCO_2 which inhibits the respiration until the PO_2 falls below 60 mm of Hg.
- The response is greater if the $PaCO_2$ is raised acutely.
- They also respond to acidaemia, respiratory oscillations of PCO_2, hyperkalemia, and drugs like Doxapram and Almitrine.
- *Central chemoreception is relatively slow process so that the fast component of the respiratory response to CO_2 is due to peripheral chemoreceptor activity.*

- **In fact the peripheral chemoreceptor *rate of response is fast enough to transduce respiratory oscillations into an oscillating nerve discharge.***
- During exercise, ventilation increases by just the right amount to excrete this CO_2 load and maintain the mean $PaCO_2$ constant.

VENTILATORY RESPONSE TO HYPOXIA

- Respiratory response to hypoxia is entirely mediated through peripheral chemoreceptors.
- *The central effect of hypoxia is depressive.*
- Acute hypoxia increases the respiratory sensitivity to CO_2.
- It is believed that in patients with chronic bronchitis or emphysema and chronic obstructive lung diseases the $PaCO_2$ is already raised to a very high level that the respiratory center has become insensitive to the stimulant effect of CO_2, and the *respiration is maintained by the hypoxic drive through the peripheral chemoreceptors.*
- That is the reason why, while instituting oxygen therapy to such patients in the acute exacerbations, regulated percentage oxygen not exceeding 30% is administered via ventimask so as to maintain the hypoxic drive of respiration. This view has been questioned now.

Hering-Breuer Reflexes

- The stretch receptors are situated in the bronchial smooth muscles, when they are stimulated by hyperinflation of lung, impulses are sent to the respiratory center via the vagus nerve, to limit further inflation (Inflation reflex).

- Similarly Hering-Breuer deflation reflex initiates inspiratory activity at very low lung volumes.
- Inflation of lungs causes reflex cessation of inspiration because the stretch receptors send impulses to the pons via the vagus nerves which inhibit the respiratory center.
- This reflex is likely to be stimulated when ventilation with large tidal volume.

Wakefulness on Respiration

- The stimulant effect of wakefulness on respiration can be ascribed tentatively to the **brain stem reticular system.** The depression of respiration on the onset of natural sleep may be due to the lack of wakefulness.

Other Drives to Breathing during Exercise

Temperature

- A rise in body temperature results in an increase in ventilation and *greatly exaggerates the respiratory response to CO_2.*
- Hypothermia causes a reduction in ventilation in excess of what would be expected on the basis of a reduced CO_2 production.

Neural Factors

The neurogenic component may originate in the *mechanoreceptors* in the joints and *the muscle spindles*; in addition there may be a contribution from other areas of brain.

Humoral Factors—Arterial pH and PCO_2

- During heavy exercise a metabolic acidosis develops, which stimulates both central and peripheral

chemoreceptors. Arterial PO_2 does not fall during exercise.

- Catecholamine levels increase during exercise which increases ventilation.
- The most important chemical factor is *potassium released* from the exercising muscles stimulate peripheral chemoreceptors.

NORMAL PROTECTIVE MECHANISMS IN RESPIRATORY SYSTEM

Mucociliary Clearance System

- This consists of the mucous and the ciliated cell of the nasal cavity of the upper airway, and the lower airway-the tracheobronchial tree.
- In the nose this functions to trap the airborne particles and transport them towards the nasopharynx. In the lower respiratory tract it moves the particle from lower down towards the glottis.
- Ciliated epithelium is present from the upper airway down to the terminal bronchioles. Each cell has about 200–275 cilia that beat upward in a coordinated, wavelike fashion at a speed of 10–20 mm/minute. Once the secretions reach the oropharynx, it is eliminated either by expectoration or by swallowing.
- Mucous comes from two sources; the surface goblet cells and the submucosal glands, which produce a mixed serous and mucous secretion. The submucosal glands respond to vagal stimulation which increases output. Vagal blocking drugs like atropine decrease the secretion and anticholinesterase drugs like neostigmine increase it.
- The normal production of mucous in the lung is about 100 ml/day.
- Immunoglobulins and enzymes are also found in the mucous blanket of the lungs. **IgA** is the principal

antibody found in the normal secretions. The deficiency of the immunoglobulins may predispose to respiratory tract infections.

- Considering the fact that the respiratory tract is in constant contact with the environment, *it is remarkable that the lower respiratory tract is almost sterile.* This is possible because of *the mucociliary clearing system, enzymes, alveolar macrophages, the pulmonary lymphatic system, and immunoglobulins.*
- *This protection is deliberately removed in an intubated ventilated patient.*
- Factors depressing the mucociliary clearing system are; ***Hyperoxia, Hypoxia, hypercapnea, inadequate humidification of inspired air, dehydration, narcotics, sedatives, alcohol, acute respiratory infection, sleep, and increasing age.***

Sigh

- All human beings sigh about 10 times per hour.
- The purpose of sigh is to counteract small airway closure, which can occur when tidal volumes are monotonous. This will open up the undereventilated alveoli.
- In earlier days, sigh was also instituted when the patient is on ventilator with about 1.5 times the tidal volume.
- Sigh should not be used with unphysiological tidal volumes 15 ml/kg.
- In modern ventilators sigh is not presented as a feature.

Cough

- This is *a cholinergic vagal reflex*; the receptors are primarily located in the upper airway.
- Mechanical, chemical or physical stimuli may initiate cough reflex.

- Cough response represents an important protective mechanism and it utilises forced expiration as the basic pattern for expelling foreign objects from the respiratory passages.
- The individual inspires, closes the glottis and vigorously contracts the expiratory muscles in a valsalva maneuver, and then suddenly opens the glottis.
- Because the air is prevented momentarily from leaving the lungs, intrapleural and intrapulmonary pressures are built to maximum levels.
- On the sudden opening of the glottis, a blast of air is forced through the trachea, effectively throwing out the foreign object or mucous deposits from the airway.
- When endotracheal tube is in the trachea, the mechanics of cough are disrupted and elimination of foreign material or mucous from the trachea cannot happen by this reflex and needs removal by suction.
- The most important problem is that, the mucociliary escalators bring the mucous from the distal part of the tracheobronchial tree towards the glottis which stops at the level of the tip of the endotracheal tube and accumulate there.
- ***With each ventilatory movement the collected secretions move in and out of the tip of the tube, eventually gets dried up and form*** *encrustations* ***at the tip of the endotracheal tube. Depending upon the amount of secretions, encrustations may severely narrow the lumen of the tip of the tube sooner or later. This results in enormous increase in resistance to breathing.***

Sneeze

- This reflex is elicited by the irritation of the mucous membrane of the nose by mechanical, physical or chemical stimuli.

- A deep inhalation is initiated followed by a violent exhalation through the nose to clear any foreign materials in the upper airway mainly in the nose.
- It is mediated by the sensory receptors of the trigeminal or olfactory nerves.

SYMBOLS USED IN RESPIRATORY PHYSIOLOGY

While discussing the physiology of respiration and mechanical ventilation, various universal symbols are used and hence it is necessary to be conversant with these symbols for easily following the discussions.

Primary Symbols

V = Gas volume
P = Gas pressure (partial pressure)
F = Fractional concentration (percentage) of dry gas
D = Diffusing capacity
R = Respiratory exchange ratio
C = Gas content in blood
Q = Blood volume
S = Saturation of hemoglobin with O_2.

Secondary Symbols for Location of Quantity

Gas Phase (Small capitals)

I = Inspired gas
E = Expired gas
A = Alveolar gas
T = Tidal gas
D = Dead space gas
B = Barometric.

Blood Phase (Small letters)

a = Arterial blood
v = Venous blood

c = Capillary blood
t = Total
s = Shunt

Examples:

- P_AO_2 = Alveolar oxygen tension (partial pressure)
- PaO_2 = Arterial oxygen tension (partial pressure)
- $PaCO_2$ = Arterial carbon dioxide tension (partial pressure)
- DO_2 = Diffusing capacity of oxygen
- Qc = Blood flow through the pulmonary capillaries
- VO_2 = Rate of oxygen consumption
- FIO_2 = Fractional inspired oxygen concentration (percentage)
- PIO_2 = Partial pressure of oxygen in inspired gas
- SaO_2 = Saturation of hemoglobin with oxygen in arterial blood.
- PB = Barometric pressure
- CaO_2 = Arterial oxygen content
- V_D = Dead space volume
- V_T = Tidal volume

Abbreviations for Lung Volumes

V_T = Tidal volume
IRV = Inspiratory reserve volume
ERV = Expiratory reserve volume
RV = Residual volume
CV = Closing volume

Abbreviations for Lung Capacities

IC = Inspiratory capacity (IRV + V_T)
VC = Vital capacity (IRV + V_T + ERV)

FRC	=	Functional residual capacity (ERV + RV)
TLC	=	Total lung capacity (IC + FRC).

It has to be noted that a capacity is always the sum of two or more volumes.

Abbreviation in Pulmonary Mechanics

C_{ST}	=	Static compliance
C_{DYN}	=	Dynamic Compliance
C_{TL}	=	Total Compliance (Chest wall + Lung)
Raw	=	Airway resistance
WOB	=	Work of Breathing
V_E	=	Volume of expired gas per unit of time (Minute ventilation).
MVV	=	Maximum voluntary ventilation
V_T	=	Tidal volume
VC	=	Vital capacity
MIP	=	Maximum inspiratory pressure
f	=	Respiratory frequency (breaths per minute)
RR	=	Respiratory rate
f : V_T	=	Ratio of respiratory frequency to tidal volume

Perfusion

Q	=	Blood flow
QT	=	Cardiac out put; also abbreviated as CO
PVR	=	Pulmonary vascular resistance
SVR	=	Systemic vascular resistance
VO_2	=	Volume of oxygen consumed per unit of time (Oxygen consumption per minute)
VCO_2	=	Volume of carbon dioxide produced per minute

Ventilation/Perfusion Relationship

V/Q = Ratio of ventilation to perfusion

Q_S/Q_T = Ratio of shunted blood to total blood flow (physiologic shunt)

V_D/V_T = Ratio of dead space to tidal volume ventilation (physiologic dead space)

In the last two chapters, the anatomy and physiology of respiratory system have been discussed briefly. Inevitably there is a little of overlapping which is necessary for following the subject better.

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CHAPTER 5

Oxygen

- *Physiological importance*
- *Availability*
- *Oxygen transfer across "alveolar capillary membrane"*
- *Transport of oxygen in blood*
- *Oxygen dissociation curve*
- *The oxygen cascade*
- *Hypoxia*
- *Oxygen toxicity*

"Oxygen remains a fascinating paradox. Essential to maintain life within a narrow spectrum of partial pressures, it is lethal at pressures outside that range."

—*Smith*

PHYSIOLOGICAL IMPORTANCE

It is quite interesting to note that, earth's atmosphere originally contained no oxygen and it was only when chemical evolution reached the stage of photosynthesis, plant like organisms began to add oxygen to the atmosphere. 'Photosynthesis' implies that the energy for the reaction is derived from light. We consume the food synthesized by the plants in this way. Therefore, we get the energy from the sun.

Within our cells this reaction is reversed so that the solar energy incorporated into the glucose molecules is released and made available for metabolic process.

$$\mathbf{C_6\,H_{12}\,O_6 + 6\,O_2 = 6\,H_2O + 6\,CO_2} + \text{Energy.}$$

Higher organisms like us are completely depending on this process of oxidation in order to derive sufficient energy from food substrates to maintain life. Oxygen is essential for this intracellular oxidation process that generates energy.

The remarkable natural balance in the atmosphere of earth is that oxygen is available at an almost constant percentage of **20.94%** (21%), possibly from ancient times when animal existence started in this world. It is one of the wonders of nature that the oxygen is provided in such a precision that its percentage in atmospheric air at sea level is constant. The atmospheric oxygen is essential and quite sufficient for sustaining life in the world.

Only in high altitudes and in certain clinical situations, additional oxygen may be required for life support. The additional oxygen has to be derived from sources such as a compressed cylinder. Even this oxygen is not derived from any artificial source, but acquired from the atmosphere and stored. *"Ex nihilo nihil fit"* meaning, *"Out of nothing, nothing comes"* suits everywhere, so also for oxygen.

Looking at history, the following noted events about oxygen are found.

- Stephen Hale in 1727, prepared oxygen.
- Priestley in 1777, showed that it is a normal constituent of air.
- Lavoisier and others in 1780 and in 1789, demonstrated that oxygen is absorbed from the lungs and after metabolism in the body eliminated as carbon dioxide and water.

AVAILABILITY

Commercial Preparation

Commercially oxygen is prepared by fractional distillation of liquid air. The composition of atmospheric air is,

- **Oxygen: 20.94%**
- **Nitrogen: 79.02%**
- **CO_2: 0.04%**

  Before liquefaction the CO_2 is removed.

Afterwards the oxygen and nitrogen are separated by evaporating the liquid air using the differences in their boiling points.

- Boiling point of oxygen: minus 182.5°C.
- Boiling point of nitrogen: minus 195.8°C.

During evaporation of liquid air the nitrogen will escape first. At 182.5°C the container will have only liquid oxygen and is evaporated. This gaseous oxygen is compressed into cylinders under 120 atmosphere pressure or 1800 lbs/sq inch (psi). (One atmosphere =15 psi). 120 × 15 = 1800 psi. The cylinders are colored black with white shoulder.

Presentation

- Oxygen cylinders.
- A central pipe line outlet which is supplied by bulk cylinders in 'manifold' room.
- Oxygen concentrator.

Oxygen is commonly presented in cylinders for medical purposes whether it is used in wards for oxygen therapy, used in ventilator therapy, or used in anesthetic machines for anesthetic purposes.

Pipeline supply also is normally fed by cylinders through 'manifold system' and only in very big hospitals with higher needs. It is fed from liquid oxygen tanks.

Cylinders

These cylinders are available in different sizes. Different countries have different nomenclature for the type of cylinders. In our country for medical purposes 12 cubic feet (AA Type), 20 cubic feet (A Type), 40 cubic feet (B Type) and D type with higher capacity are available.

- 12 cubic feet. (AA Type) and 20 cubic feet (A Type) are fitted with ***"Flush valves"*** for fixing on anesthetic machines. In normal way, it can be used in anesthetic machines only.
- 40 cubic feet (B Type) is fitted with ***"Bull nose valve"*** is meant for oxygen therapy in wards.
- Bulk cylinders (D Type) are fitted with ***"Bull nose valve".*** These are used in manifold room for feeding the central pipe line supply.

When the weekly requirement of the hospital is above 50,000 cubic feet, ***'Liquid oxygen"*** supply is used.

OXYGEN CONCENTRATOR

There is another way of retrieving oxygen from the atmospheric air by using an oxygen concentrator. This equipment separates oxygen from atmospheric air by preferential absorption of nitrogen on 'Zeolites' (crystalline aluminosilicates). The "Zeolite sieve" traps almost all the nitrogen from the air and delivers more than 90% of oxygen. The domestic model can give an output of 95% oxygen at a flow rate of 2 L/min with harmless impurities such as argon.

Oxygen concentrators are available for hospital uses also, this is not the common method of getting the supply of oxygen.

The percentage of oxygen (and other gases) in air and respiratory tract

	Atmospheric air	*Alveolar air*	*Expired air*
Oxygen:	20.94%	14.2%	16.3%
CO_2	0.04%	5.5%	4.0%
Nitrogen:	79.02%	80.3%	79.7%

The oxygen from atmospheric air reaches the alveoli during inspiration, diffuses through alveolar capillary membrane into the blood, and transported to the cells for metabolism, more precisely to the level of mitochondria. In this process there is a progressive stepwise fall in the partial pressure of oxygen and by the time it reaches mitochondria; inside the cells the partial pressure (PO_2) is only 4 mm Hg. (see oxygen cascade)

PRESSURE GRADIENT OF OXYGEN FROM ALVEOLI TO BLOOD

- The alveolus at body temperature of 37°C has a constant partial pressure of water vapour constituting 47 mm of Hg.
- So in atmospheric pressure of 760 mm Hg, the ***total pressure of gases in the alveoli*** can be only ***713 mm Hg***. (760 – 47 = 713).
- As the atmospheric air containing **21% oxygen**, mixes with the expired air in the process of reaching alveoli, the oxygen gets diluted and the percentage drops to **14.2%** *in the alveoli.*
- The partial pressure of ***oxygen in the alveolus*** of a person breathing atmospheric air is (14.2% of 713 mm Hg = 101.2 mm Hg) approximately ***103 mm Hg.***
- The partial pressure of oxygen in the venous blood reaching the lung is 40 mm Hg. Hence, a pressure gradient of 60 mm Hg is present which is adequate for the oxygen to diffuse into the blood. *This is the force that drives the oxygen across the alveolar capillary membrane into the blood.*
- When blood passes through the pulmonary capillaries, this pressure gradient, causes rapid diffusion of oxygen to reach the equilibrium of 100 mm Hg. (103 mm Hg

equilibrium will not be reached normally because of the ventilation perfusion mismatch).

Oxygen Transfer Across "Alveolar Capillary Membrane"

- The blood passes through the pulmonary capillaries for 0.75 second, but the equilibrium is reached in about 0.25 seconds. The remaining 0.5 seconds is the reserve available.
- In hyperdynamic circulation as in severe exercise, the blood passes through the capillaries faster and even if the time is reduced to 0.25 seconds, adequate equilibrium is reached.
- If the patient is breathing 100% oxygen from a mask, the nitrogen in the lungs is removed gradually and the lungs are fully filled with 100% oxygen after approximately 3 minutes time (denitrogenation of lung). The partial pressure of oxygen in alveoli is 673 mm Hg.

❖ Atmospheric pressure:	760 mm Hg.
❖ Saturated water vapour at 37°C:	47 mm Hg.
	713 mm Hg
❖ Alveolar CO_2 (5.5%):	40 mm Hg.
❖ Alveolar oxygen:	673 mm Hg.

THE PARTIAL PRESSURES OF OXYGEN

It will be interesting to note how the partial pressure of oxygen from atmospheric air is transferred and adjusted in the respiratory tract in such a way that by the time it reaches the arterial blood it is sufficient to deliver enough oxygen to the tissues. Table 5.1 shows the partial pressure

of oxygen getting reduced gradually as it reaches the tissue level.

Table 5.1: Partial pressures of various gases

	Atmospheric air	*Expired air*	*Alveolar air*	*Arterial blood*	*Venous blood*
Oxygen	159.0	150.0	103.	96	40
CO_2	0.3	0.3	40	40	46
Nitrogen	600	563	573		
H_2O	5	47	47		

The *oxygen cascade* will explain how the partial pressures are decreased in a form of cascade, so that by the time the mitochondria are reached, the partial pressure of oxygen is only 4 mm Hg (see Page 114).

METABOLIC REQUIREMENT OF OXYGEN

- Basal metabolic requirement of oxygen is about 250 ml/min.
- Arterio-venous oxygen difference is about 5 to 6 ml/100 ml of blood, which means that when 100 ml of blod perfuses the tissues, it gives out 5 to 6 ml of oxygen for metabolism.
- Therefore, 5000 ml of blood (cardiac output) gives off 250 ml of oxygen/min.

Each organ according to its ***metabolic requirements*** takes its ***share*** out of the ***250 ml of oxygen.***

Organ	*Weight*	*Blood flow*	*Oxygen utilization*
Brain	1500 gm	50 ml/100 gm	**52 ml/**min (3.5 ml/100 gm)
Liver	3000 gm	1500 ml/min	**60 ml/**min (300 ml – Arterial)
Kidney	300 gm	1200 ml /min	**18 ml/**min (25% cardiac output)
Heart	300 gm	250 ml /min	**40 ml/**min (15% cardiac output)

Looking at the table, it is clearly understood that,

- The liver needs highest amount of oxygen but is supplied by relatively hypoxic blood, 80% from portal vein.
- The heart is a relatively poorly perfused organ. It needs 40 ml of oxygen per minute but is perfused with only 250 ml of blood/min.

Oxygen Expenditure for Work of Breathing

The energy required for ventilating the lung. It is simplified as the expenditure of oxygen for doing the work.

- 0.5 ml of oxygen/1 litre of ventilation.
- 4 ml of oxygen/8 litres of ventilation. (minute volume)
- It is 1.5% of total oxygen consumption/min.

Transport of Oxygen in Blood

In the *alveolar air the partial pressure* of oxygen is approximately **103 mm** Hg whereas it is **only 40 mm Hg** *in the venous blood* entering the pulmonary capillaries. This pressure *gradient of about 60 mm Hg* is sufficient to drive oxygen rapidly across the alveolar capillary membrane. On entering the blood stream, the oxygen becomes dissolved in the plasma before finally uniting with hemoglobin to form ***oxyhemoglobin*** for its transport to tissues.

Oxygen Dissolved in Plasma

Only a very small proportion of the total oxygen carried in the blood, namely ***0.3 ml of oxygen per 100 ml of blood*** is physically dissolved in the plasma.

- Nevertheless this small quantity is of vital importance for; it alone reflects the tension of oxygen in blood (PaO_2).

- This acts as a pathway for the oxygen to reach the hemoglobin that is contained in RBCs.
- When the blood reaches the tissues, it is this small quantity that is first transferred to the cells, while its place is rapidly taken by more oxygen liberated from hemoglobin.

Oxyhemoglobin

- Hemoglobin consists of the protein 'globin' joined with the pigment 'hem' which is an iron containing porphyrin.
- The porphyrin nucleus consists of four pyrrol rings joined together by four methine bridges. The iron in the hemoglobin has a loose bond available for the union with oxygen to form oxyhemoglobin.
- The union of oxygen with hemoglobin is very easily formed. In the same way oxygen is released from oxyhemoglobin as easily as it is formed.
- In the arterial blood the ***PO_2 is 100 mm Hg*** and consequently hemoglobin is almost completely in its oxygenated form. In the ***tissue level the PO_2 drops to 40 mm Hg*** and the hemoglobin is now forced to give up some of the oxygen it is holding.
- ***1 gm of hemoglobin*** when ***fully saturated*** (100% saturation) can combine with ***1.39 ml of oxygen.***
- When a healthy individual has hemoglobin level of 14.5 gm/100 ml of blood, this 100 ml of blood can theoretically carry about 20 ml of oxygen.
- Complete equilibration between the alveolar and arterial tension is never reached (because of normal physiological ventilation-perfusion mismatch), so that arterial blood under normal conditions is seldom more than 95% saturated, with an oxygen tension of 100 mm Hg.

- Thus 15 gm (it is rarely seen) of hemoglobin present in 100 ml of blood can carry approximately 19 ml of oxygen.
- It will be seen, therefore, that ***every 100 ml of*** blood passing through the lungs ***takes up 5 ml of oxygen***.
- The venous blood reaching the lungs has 70% saturation and holds 14 ml of oxygen/100 ml of blood. So arterial – venous 14 ml = 5 ml.
- Equally, when the blood reaches the periphery, the tissues remove about 5 ml of oxygen from 100 ml blood. (i.e. one third or one quarter of the total it holds).

Now it is proper to discuss about the oxygen dissociation curve and its significance in clinical practice.

OXYGEN DISSOCIATION CURVE

When the percentage saturation of oxygen in hemoglobin in relation to the partial pressure to which it is exposed is plotted in a graph, the saturation has no linear relationship to the increasing partial pressures.

- The saturation of oxygen in hemoglobin (whole blood with normal CO_2 content at body temperature) against the various partial pressures is plotted in a graph; it forms a characteristic "S" shaped curve that is known as *Oxygen Dissociation Curve (ODC)* (Fig. 5.1).

  This peculiar "S" shaped curve has certain advantages to the patient.
- The tension, (the partial pressure) PaO_2 can fall from 95 mm Hg to 80 mm Hg as in the case of slight respiratory obstruction, yet the hemoglobin in the arterial blood virtually remain fully saturated, say 97%.
- In fact the tension has to fall to 60 mm Hg before cyanosis is visible; even then the arterial saturation is around 80%.

- At the beginning of the curve, when the PO_2 is low in the tissues, the hemoglobin readily gives up oxygen continuously.

The percentage saturation of hemoglobin largely depends up on the tension of oxygen in the blood. When there is an increase in the tension, there is an increase in the saturation. As we have seen earlier, this relationship is not linear as it is with the solution of oxygen in plasma.

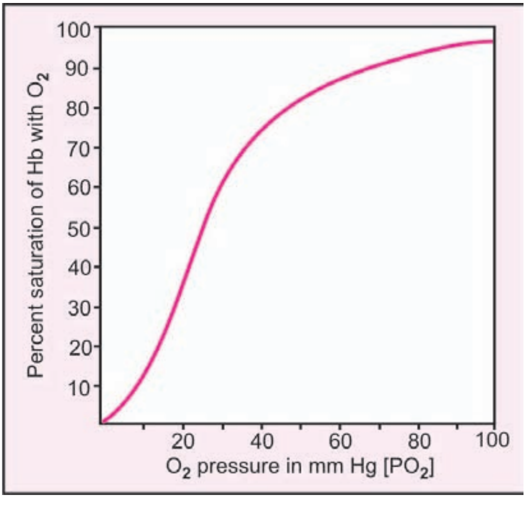

Fig. 5.1: Oxygen dissociation curve (ODC) of human blood

Oxygen tension	*Solution in plasma*	*Saturation of hemoglobin*
40 mmHg	0.12 ml	75%
80 mmHg	0.24 ml	95%
100 mmHg	0.3 ml	97%

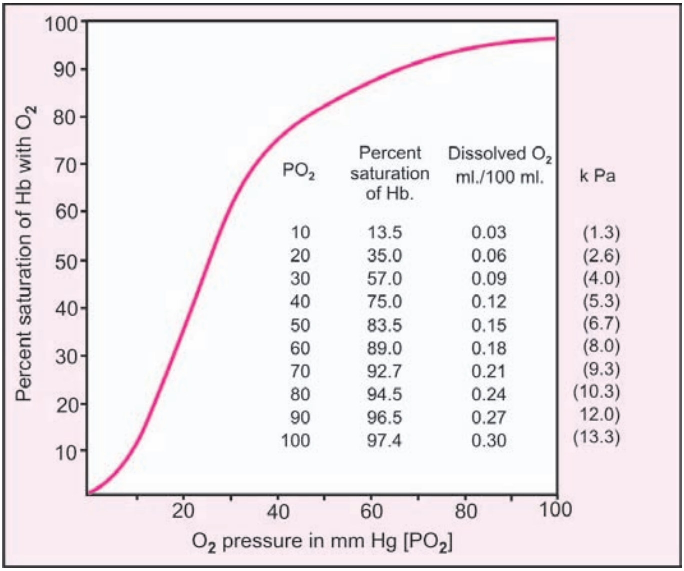

Fig. 5.2: Oxygen dissociation curve showing various partial pressures and saturation

- ❖ Note various partial pressures and the corresponding saturation of hemoglobin causing the “S” shape of the curve.
- ❖ Note that solution in plasma increases in a linear relationship to the partial pressures to which it is exposed.

- Hence, the amount of gas taken up by plasma or blood varies according to the tension of the gas to which it is exposed.
- Taking oxygen as an example, the amount that is *dissolved in plasma is directly proportional to the partial pressure, i.e. there is a linear relationship*. For example, breathing atmospheric air the oxygen tension in the arterial blood is 100 mm Hg and 0.3 ml oxygen is dissolved in plasma in 100 ml of blood (Fig. 5.2).

- When the same person breathes **100% oxygen** from an anesthetic machine, the tension of oxygen is (760 mm – 47 mm water vapour) approximately 7 times more and so the **oxygen dissolved in plasma is 0.3 × 7 = 2.1 ml**. In this way there is a theoretical possibility of carrying 21 ml of oxygen in plasma by using hyperbaric oxygen without the need for hemoglobin.
- On the other hand, the iron in the hemoglobin molecule has a remarkably different property of combining with oxygen to form oxyhemoglobin as seen in oxygen dissociation curve.
- When this curve is analysed, it shows the distinct advantage that *the tension of oxygen can fall considerably yet the oxygen saturation remains relatively high.*
- It is to be noted that at a tension of 100 mm Hg the saturation is 95 percent and at 40 mm Hg, it is still 75 percent (Fig. 5.2).
- This curve provides ideal conditions for **the uptake** and **unloading** of **oxygen.**
- When the oxygen tension is increased to 760 mm Hg by breathing 100% oxygen, the saturation can increase from 95 to a maximum of 100%. That is to say a point is soon reached where any increase in tension is not followed by an equivalent rise in saturation.
- Various physiological factors can shift the curve either to the right or to the left.
- It is evident that ***the shift to the right is advantageous,*** as it allows the hemoglobin to *easily part with oxygen* so, that delivery to tissues continues even at low saturation relatively preventing hypoxia at tissue level.
- ***Shift to the left is disadvantageous***. This means that hemoglobin has high affinity for oxygen and does not easily part with its oxygen so that even at higher saturation there is ***minimal unloading*** of oxygen at tissue level causing tissue hypoxia.

In clinical practice, to prevent tissue hypoxia, factors that shift the curve to the left are looked for and must be avoided. Hence during mechanical ventilation. It is essential to see that the factors that cause shift of ODC are carefully avoided in clinical practice.

The Important Factors that Shift the ODC

Carbon Dioxide Tension

A rise in carbon dioxide tension tends to *'shift the curve to the right'*. This creates an interesting phenomenon. In the tissues the carbon dioxide tension is high so the shift to right occurs, the hemoglobin can more readily give up its oxygen whereas in *the pulmonary venous blood the carbon dioxide tension* is low and therefore curve is *'shifted to left'* and *the uptake* of oxygen is aided.

The pH of the Blood

Acidemia shifts the ODC to the right and alkalemia to the left (Figs 5.3A and B).

Stored Blood

2, 3 Diphosphoglycerate (2, 3 DPG) concentration within the red cell modifies the curve. A high concentration causes the curve shifted to right. Stored blood has low levels of 2, 3 DPG depending up on the number of days of storage. It takes more than 48 hours (2 to 3 days) for 2,3 DPG level to recover to normal.

Temperature

A fall in temperature shifts the curve to be shifted to **the left**. The shift causes less oxygen delivery to the tissues; incidentally hypothermia also reduces the oxygen demand.

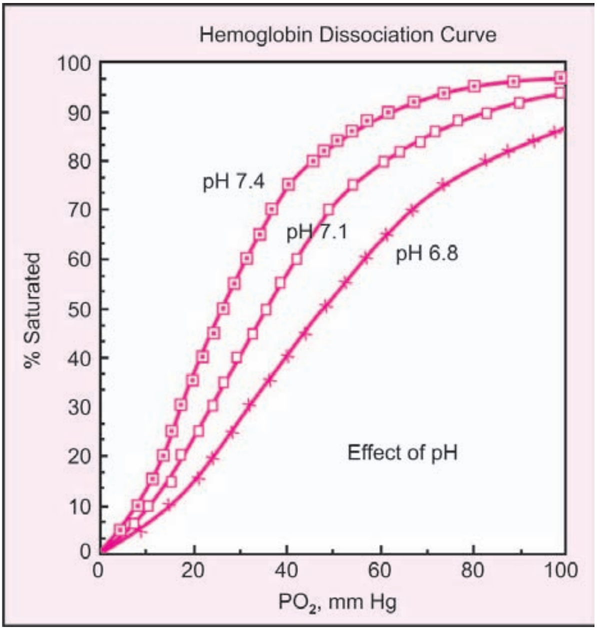

Fig. 5.3A: ODC shifted to right by acidemia

Clinical Significance

Discussion

When a patient ventilated with atmospheric air breaths 21% oxygen and 79% N_2, the effect of ventilation perfusion maldistribution is sufficient to reduce the oxygen tension of the arterial blood. Fortunately the shape of the oxygen dissociation curve for blood is so arranged that despite a significant drop in the tension the saturation level is minimally affected. However, there are clinical situations when the tension may fall to a level where a further small reduction would lead to a dangerous drop in saturation.

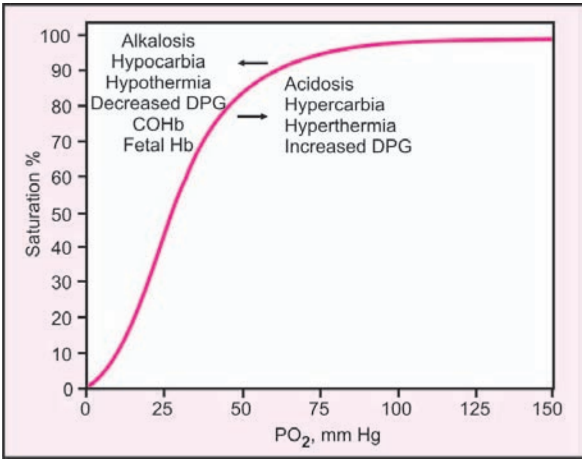

Fig. 5.3B: Various factors that cause shift of ODC to the right or to the left

These facts can be best emphasised by referring to the analogy of the child playing on a cliff top (Fig. 5.4). *The sea, the cliff top, and the grass field on the top represent the oxygen dissociation curve.* The conscious subject breathing air (21 percent oxygen) is represented by the child at **point A**. It will be observed that fairly wide movements to the right or left will lead to only a very small change in the oxygen saturation.

The patient on ventilator or those in the immediate post operative period under the effect of opioid analgesic drugs are quite different. Now the child has moved to the **point B**. Any movement to the left (as for example, with hypoventilation or mild respiratory obstruction) can lead to a perilous descent towards desaturation. If, however, such patients are given extra oxygen, they are rapidly transported to the **safety zone (point A)** enjoyed by conscious subjects.

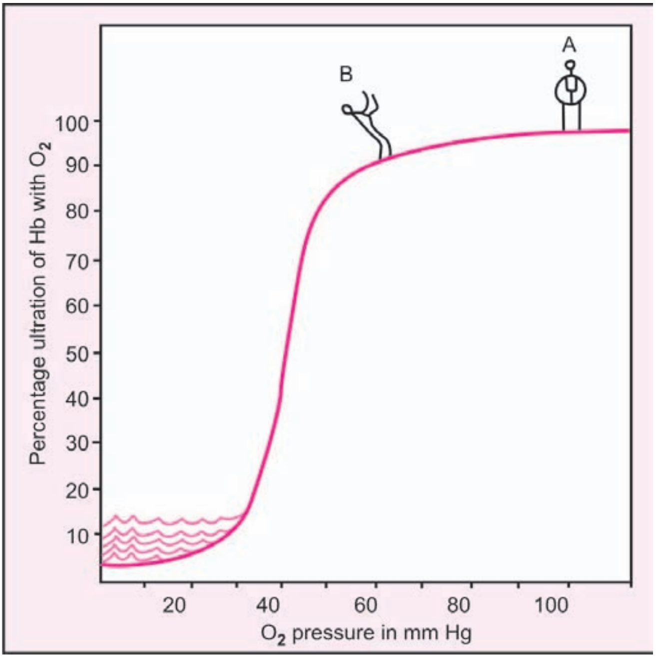

Fig. 5.4: ODC with an analogy of a child playing on a cliff

It is recommended, therefore, that at least 33 percent oxygen should be the minimum inspired concentration for the patients on mechanical ventilation. The reason is quite obvious, as discussed earlier, instituting mechanical ventilation itself increases the physiological dead space and the chance for hypoxemia. Ideally a higher percentage is likely to combat the ventilation perfusion mismatch in a patient mechanically ventilated.

HYPOXIA

*The failure of the tissues to receive adequate quantities of oxygen is variously described as anoxia, **hypoxia** or **oxygen lack**.*

The lack of oxygen represents a serious hazard to the tissues and has been aptly described by Haldane as causing *"not only stoppage of the machine, but also ruin the supposed machinery".*

Hypoxia due to any cause is universally one of the primary causes of morbidity or mortality in critically ill patients.

Under ordinary conditions the body has certain regulatory mechanisms which prevent the tissues from suffering oxygen deprivation, but in critically ill patients oxygen lack may become a factor of prime importance.

Oxygen is essential for cells metabolism and to realise how it can affect cell metabolism when oxygen lack occurs, the following discussion may be useful.

As discussed in the beginning of this chapter we consume the food synthesized by the plants. Within our cells, the solar energy incorporated in the glucose molecules is released by oxidation and made available for cell process.

$$C_6H_{12}O_6 + 6\,O_2 = 6\,H_2O + 6\,CO_2 + \text{Energy.}$$

Higher organisms like us are completely dependent on this process of oxidation in order to derive sufficient energy form food substrates to maintain life. Some energy can be obtained from glucose in the absence of oxygen (anaerobic), although in terms of adenosine triphosphate (ATP) production. Anaerobic metabolism is only **1/19**th as efficient as aerobic metabolism.

The energy is stored in the form of a high energy phosphate bond in the ATP molecule. When the energy is

required, ATP is broken down to adenosine diphosphate (ADP) and phosphate. Lactic acid accumulation as a result of anaerobic metabolism will lead to a metabolic acidosis, although when oxygen becomes available the lactate can be metabolized to CO_2 and H_2O with further energy release and ATP formation.

The Oxygen Cascade

The following discussion about the oxygen cascade will explain how *a drop in* PaO_2 will eventually affect the partial pressure in cellular level, particularly in mitochondria is likely to cause severe damage to the function of cells.

The oxygen cascade shows the stages in which the PO_2 falls in steps as follows;

- PO_2 in Dry Atmospheric air ——**158 mm Hg**
- PO_2 in Alveoli ——**105 mm Hg**
- PO_2 in Arterial blood—— **100 mm Hg**
- PO_2 in Capillary blood —— **40 mm Hg**
- PO_2 in Mitochondria—— **4 mm Hg**
- PO_2 drops through the cytoplasm to the mitochondria from 40 mm Hg to 4 mm Hg.

PO_2 drops in stages from 158 mm Hg in dry air to the low levels in the mitochondria. Alveolar PO_2 in young fit adult is about 105 mm Hg. The first drop occurs because of humidification. Fully saturated air in the alveoli at 37° C has a PO_2 of 105 mm Hg.

Therefore, in the alveolus it is about 105 mm Hg. The alveolar-arterial oxygen difference is in the order of a few mm Hg. The PO_2 then falls progressively from the arterial end to the venous end of capillaries and there is

variable gradient from the capillary blood to the cells. The PO_2 is the lowest in mitochondria. *Most of the oxygen is utilized in the mitochondria and if the level of PO_2 falls* ***below 1 to 2 mm Hg****, the so called* ***Pasteur point,*** *then aerobic metabolism stops.*

Classification of Hypoxia

Based on the causes, the hypoxia is classified into four types. They are,

- ***Hypoxic hypoxia***
- ***Anemic hypoxia.***
- ***Stagnant hypoxia.***
- ***Histotoxic hypoxia***.

It is necessary to understand the mechanisms in the four types of hypoxia, as it is possible to correct three among the four types of hypoxia by simple means thereby improving the cellular oxygenation. It is absolutely essential in a critically patient for a better outcome. *Many times there may be a combination of more than one type hypoxia in a patient.*

Hypoxic Hypoxia

Simply it means less oxygen reaches the blood and so less delivery at the tissue level. It is otherwise called as Hypoxemia. This is very commonly encountered during clinical practice. There are various causes for this hypoxic hypoxia. They are,

Reduced partial pressure of oxygen in the inspired gas (Low P_AO_2)

This can be due to a fall in F_IO_2 (fractional concentration of oxygen in the inspired gas is low) or a fall in barometric pressure as in high altitudes. In other words, if the patient

is breathing a hypoxic mixture, naturally less oxygen reaches the alveoli and so the blood. Similarly at the top of Mount Everest at 29028 ft height, the barometric pressure is only 236 mm Hg compared to 760 mm Hg at sea level. Therefore, at this height the partial pressure of oxygen PIO_2 is only 40 mm. At this level hypoxia inevitably occurs if additional oxygen is not added to inspired air by mask.

Diffusion hypoxia or finks effect

This condition is related to anesthesia with N_2O. When breathing N_2O relatively large amount of this gas replaces the less soluble nitrogen in the body fluids. N_2O is 34 times more soluble in blood than N_2. When the anesthetic is stopped, this nitrous oxide diffuses from the body tissues via venous blood into the alveolar gas and nitrogen now being breathed, diffuses back into the tissues. Because the solubility of nitrous oxide much greater than that of nitrogen a relatively small amount of nitrogen passes from the alveolar gas to restore the tissue PN_2 but a much larger amount of N_2O passes from the tissues to the alveoli, where it dilutes the alveolar gas and so reduces the PO_2. The opposite occurs during induction and has a concentrating effect on the other gases including oxygen.

Postoperative diffusion hypoxia is usually brief and in healthy fit individuals it is insignificant, but in severely ill, high-risk patients it may cause damage.

Reduced alveolar ventilation

This may be due to varied reasons which have been discussed in earlier chapters.

Reduced diffusing capacity

The term ***alveolar/capillary block*** is used to describe impairment of oxygen diffusion from the alveolar gas across the alveolar membrane and pulmonary capillary wall into

the pulmonary capillary blood as a result of thickening of alveolar-capillary membrane.

Anemic Hypoxia

In this form of hypoxia, *the oxygen content of the blood is reduced*, although the arterial partial pressure of oxygen is normal. If the total hemoglobin is less than normal as in ***anemia*** or if part of the hemoglobin is unavailable for oxygen transport as in ***carbon monoxide poisoning, methemoglobinaemia or sulphemoglobinemia*** then anemic hypoxia will result.

Anemia

In anemia, because the hemoglobin is less the oxygen content of the blood is reduced.

Carbon monoxide poisoning

Carbon monoxide combines with hemoglobin to form carboxyhemoglobin, so that this fraction of hemoglobin is not available for oxygen transport. A relatively low concentration of carbon monoxide can form a substantial amount of carboxyhemoglobin because ***the affinity of hemoglobin for CO is about 250 times its affinity for oxygen.*** Incidentally CO causes *a shift of oxygen dissociation curve to the left, making offload of oxygen to the tissues difficult.*

Methemoglobinemia and Sulphemoglobinaemia

Characteristically cyanosis (due to the presence of either methemoglobin or sulphemoglobin in the blood) is unaccompanied by evidence of any cardiac or respiratory abnormalities. Methemoglobinemia is commoner and may be congenital or acquired. It may be caused by many chemicals like nitrites, nitrates, phenacetin, acetanilide, sulphanilamide, and prilocaine. *These hemoglobins cannot*

transport oxygen and they also shift the oxygen dissociation curve to the left.

Methemoglobinemia gives the mucous membrane *a chocolate–brown* appearance whereas sulphemoglobinemia produces a *leaden-blue color*.

Stagnant Hypoxia

This occurs because of reduced tissue perfusion which may be either general or local.

- General hypo perfusion is due to ***a low cardiac out put.***
- Local hypo perfusion may be due to an ***arterial occlusion,*** for example by atheroma, embolism, trauma, and vasoconstriction or to ***venous obstruction.***

Histotoxic Hypoxia

- In the mitochondria food substrates are oxidized by nicotinamide adenine dineucleotide (NAD) which removes hydrogen. The hydrogen is passed down an enzyme chain containing several cytochromes and finally combines with oxygen to form water. Poisoning of this enzyme system means that the cells are unable to use the oxygen being delivered to them and aerobic metabolism therefore stops. A small amount of ATP can still be formed as a result of anaerobic metabolism. Reduced production of CO_2 and stimulation of ventilation mediated by chemoreceptors produce a fall in arterial PCO_2; mixed venous PO_2 rises because the oxygen consumption has fallen.
- Sodium nitroprusside contains a cyanide radical, so overdose of this drug can cause histotoxic hypoxia due to poisoning of intracellular enzymes.

Cyanosis

- ***"Cyanosis*** and ***Hypoxia*** are ***not synonymous".*** There can be hypoxia without cyanosis and cyanosis without hypoxia.
- This term cyanosis refers to the bluish discoloration of skin and mucus membranes which occur if there is more than 5 gm/100 ml of reduced hemoglobin in the blood passing through the capillaries.
- When the absolute amount of reduced hemoglobin in the at least 5 gm%, cyanosis will manifest.
- When the patient is severely anemic and total hemoglobin percent is only around 7 gm%, then it is evident that even with severe hypoxia there may not be any cyanosis as the total amount of reduced hemoglobin is very much less than 5 gm even in severe hypoxia. ***This is hypoxia without cyanosis.***
- With a pulse oximeter, a saturation of 95 to 100% is normal. A value less than 95 to 90% is considered as mild hypoxia, from 90 to 85% is considered as moderate hypoxia and anything below 85 is considered as severe hypoxia and is physiologically damaging.
- A value less than 70% carries no meaning, as the calibration of oximeters are done with healthy volunteers and it is unwise to bring any individual to a desaturation below 70% and is unsafe and so these figures are not calibrated values.
- In fact when a ***patient manifests cyanosis,*** the arterial ***oxygen saturation*** has already ***fallen to 80% or lower,*** which means the hypoxia, is severe enough to cause serious damage to brain unless immediately corrected.
- In polycythemia where the hemoglobin% is more than 23 gm, the reduced hemoglobin may be more than 7 gm and cyanosis may manifest, but there may not be any hypoxia. ***This is cyanosis without hypoxia.***

Cyanosis may be ***central*** or ***peripheral.***

- Central cyanosis, which is best detected by looking at the mucosa of lips, implies arterial desaturation.
- Peripheral cyanosis may occur because there is central cyanosis, or it may occur alone due to poor peripheral blood flow. It is best detected by looking at the nail beds. If these are blue but lips are not, then the cyanosis is peripheral. *Detection of either form of cyanosis is prone for considerable observer errors.*

Effects of Hypoxia

- When we consider the physiological response to hypoxia, the catalogue of responses is large and *depends on the speed of occurrence and the severity.*
- The responses may be **Direct** or **Indirect.**
- The direct effects are principally on cardiorespiratory systems, whilst the secondary effects are those due to failure within organs such as *brain, liver, kidney*, when the *oxygen tension falls below a critical level.*

Direct Effects

Cardiovascular system

- Heart is an organ with high oxygen consumption of **8** to **10 ml O_2/100 gm/min** even at rest and thus it is relatively hypoxic.
- The normal response to hypoxia is a tachycardia.
- Systemic vascular resistance is reduced.
- Cardiac out put is increased.
- Peripheral chemoreceptor stimulation resulting in increase in sympathetic activity and catecholamine release.
- Cerebral vasodilatation.

These will have immense ill effect in a patient with compromised cardiovascular system stabilized by drugs.

Respiratory system

- Ventilation is increased.
- Pulmonary vascular resistance is increased and may increase the right ventricular pressure.

Metabolism

- Aerobic metabolism is reduced.
- Anaerobic metabolism starts, that leads onto metabolic acidosis.
- Organ failure; hypoxia for a fairly prolonged time may further damage the compromised organs having borderline function leading on to failure of the organ.

Indirect Effect on Other Organs

- **Brain** is the first organ to be affected by hypoxia, as there is no anaerobic metabolism in neurons.
- A normal person loses consciousness when the arterial saturation falls to 50% or below. At this point the PaO_2 may be around 26 mm Hg.
- **Liver** is another organ affected much by hypoxia. Hepatic cells arranged in lobules with centrilobular (portal blood supply) and peripheral (systemic blood supply) show a characteristic pattern of damage in hypoxic conditions.
- The centrilobular cells exhibit the first changes, being most remote from the systemic blood supply. In acute hypoxia, centrilobular necrosis is seen whilst, in more chronic hypoxic states, fibrosis develops.
- The effect of hypoxia on kidney is largely due to vascular reactions and intra renal redistribution of blood flow. Inevitably depending on the duration and severity of hypoxia, renal oedema is caused.

Postoperative Hypoxia

The need to discuss the postoperative hypoxia in this chapter is because, it has been established that in almost all patients post operative hypoxemia is an inevitable consequence of any type of anesthesia, the precise cause could not be identified and many factors have contributed for the problem.

In all the cases of postoperative hypoxemia, the $PaCO_2$ level had been within normal limits, eliminating hypoventilation as the cause. The need to assess these patients carefully because, though majority of them do well after an initial set back, in a very few of them the condition may progress to a level that may need ventilatory support.

- It has been proved beyond doubt that, the PaO_2 decreases after general anesthesia, even when respiratory function appears to be normal. There are several reasons for that which may be discussed briefly.
- The ***hypoxic hypoxia, anemic hypoxia and stagnant hypoxia*** all may be there. The important consideration is the oxygen flux and that must match the tissue oxygen requirement *(see page 77)*.
- The oxygen requirement is commonly increased by factors like shivering, pyrexia, pain, etc.

The Factors that may Cause Postoperative Hypoxia

Diffusion hypoxia or Fink effect which has been discussed earlier may occur in the immediate postoperative period. It has been dealt with earlier in hypoxic hypoxia.

Ventilation perfusion mismatch

- Anesthesia produces a reduction in functional residual capacity (FRC), the consequences of this is likely to be

more serious in the elderly and patients who are fat or smokers.

- It has been established that upper abdominal surgeries cause a reduction of FRC by 30%.
- Hypoxemia is caused by the ventilation perfusion mismatch that is inevitable during any anesthesia and more with general anesthesia.
- This can be corrected by oxygen administration which may be sometime needed for a few days.

Reduced cardiac output

- A reduced cardiac output will reduce the oxygen flux, which may be insufficient to meet the patient's oxygen demand especially if he is shivering, as shivering is likely to increase the oxygen demand even up to 300%.
- Hence, any patient who shivers in post operative period ***is in hypoxia of variable degree*** and needs oxygen supplementation. This situation is made worse if the patient is anemic.

Hypoventilation

Drugs

- Most anesthetic drugs depress the ventilation. If the dose has been judged correctly it is unlikely that a dangerous hypoventilation will result from this alone, but *even the so called short-acting opiates like Fentanyl can cause respiratory depression lasting several hours.*
- Inadequately reversed muscle relaxant drugs used during anesthesia, although the residual paralysis usually needs to be fairly gross for hypoventilation to result.

Obstruction

- Partial respiratory obstruction in the postoperative period is often not recognized immediately. This aspect has been discussed in detail in the chapter on "Maintenance of airway"

Pain

- In some patients, for example those who have had upper abdominal surgery, pain may be preventing them from breathing adequately and deeply.

Diaphragmatic Splinting

- Elevation of the diaphragm due to abdominal distention or a tight abdominal bandage, strapping, etc. will also contribute for hypoventilation.

Intraoperative Hyperventilation

- Many patients are hyperventilated during operations. When spontaneous ventilation is restored there may be *considerable total body deficit of* CO_2. Reduction of CO_2 elimination by hypoventilation during the early postoperative period allows this deficit to be made up.
- Increasing the inspired oxygen concentration by using a 4 liters/minute flow of oxygen into a light weight mask will prevent hypoxia occurring in most patients.

OXYGEN TOXICITY

"Oxygen is lethal at pressures outside a range"

Chronic Pulmonary Toxicity

- Prolonged high concentrations of oxygen are known to damage the lungs. Toxicity is dependent both on *the partial pressure* of oxygen in the inspired gas and the *duration of exposure.*
- *Alveolar oxygen tension* rather than arterial tension is most important in the development of oxygen toxicity.
- It may be caused when *concentrations over 60%* are inhaled for prolonged periods at atmospheric pressure.
- This is *probably due to inactivation of surfactant* and damage to the pulmonary epithelium by depression of mucociliary transport and depression of phagocytosis.
- Clinical manifestations in an awake normal patient may include substernal distress, reduction in vital capacity, paresthesia, joint pains, anorexia, nausea, vomiting, contracted visual fields, bronchitis and atelectasis and mental changes.
- X-ray chest may reveal bilateral patchy opacities, spreading to the whole of the lung fields, with increase in alveolar-arterial oxygen PO_2 difference, so that despite high inspired oxygen, PaO_2 may be low.
- These changes are suggested to be due to;
  1. Airway closure leading to atelectasis in the absence of nitrogen.
  2. Loss of surfactant.

Mechanism of Toxicity

Molecular oxygen (O_2) is unusual in that each atom has unpaired electrons in its outer (2P) shell. This gives the molecule a paramagnetic property that allows precise measurements of oxygen. Notably, internal rearrangement of these electrons or their interaction with other atoms (iron)

or molecules (xanthine) can produce potentially toxic chemical species.

Oxygen toxicity is thought to be due to intracellular generation of highly reactive O_2 metabolites (free radicals) such as s*uperoxide* and *activated hydroxyl ions*, *singlet* O_2, and *hydrogen peroxide*. A high concentration of O_2 increases the likelihood of generating toxic species. These metabolites are cytotoxic because they readily react with cellular DNA, sulfhydryl proteins, and lipids.

Two cellular enzymes, superoxide dismutase and catalase provide some protection by sequentially converting superoxide first to hydrogen peroxide and then to water.

Additional protection may be provided by antioxidants and free radical scavengers such as glutathione peroxidase, ascorbic acid (Vitamin C), alpha- tocopherol (Vitamin E), acetylcysteine, and possibly mannitol. However, clinical evidence supporting the use of these agents in preventing pulmonary toxicity is lacking.

Oxygen mediated injury of the alveolar-capillary membrane produces a syndrome that is pathologically and clinically indistinguishable from ARDS. Pulmonary capillary permeability increases and the membrane thicken as type I alveolar cells decrease and type II proliferate. Tracheobronchitis may also be present initially in some patients.

Pulmonary O_2 toxicity in newborn infants is manifested as bronchopulmonary dysplasia.

Retrolental Fibroplasia

Oxygen therapy in neonates with immature retina can lead to disorganized vascular proliferations and fibrosis, retinal

detachment, and eventual blindness. Neonates of less than 36 weeks gestational age are at greater risk, but even those up to 44 weeks gestational age may be at risk.

In contrast to pulmonary toxicity, *retrolental fibroplasia correlates* better with *arterial tension than with alveolar* O_2. Arterial O_2 tensions below 140 mm Hg are generally considered safe.

Acute Toxicity

- In normal healthy persons, inspiration of 100% oxygen may be harmful if continued for more than a few hours, where as in general, 40% can be inhaled indefinitely, with out ill effects.
- The highest safe concentration is not known, and anyway it will vary depending on the individual, the pre-existing pulmonary pathology, and the duration of exposure.
- In intensive care, it is customary to use the lowest level of FIO_2 which produces an acceptable level of PaO_2, and patients should not be ventilated with high concentrations of oxygen for prolonged periods unless the alternative is dangerous hypoxia.
- The crucial factor may be the level of the arterial PaO_2.
- Free oxygen radicals are proposed as the causative factor in alveolar damage in these circumstances, by inactivating the antiprotease alpha 1-antitrypsin of alveolar cells.
- Leucocytes activated by complement, then flood the area giving the typical picture of leucoaggregates and intra-alveolar hemorrhage and exudation.

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CHAPTER 6

Mechanics of Respiration

- *Respiratory apparatus and muscles of respiration*
- *Normal lung movements*
- *Normal movements of respiration*
- *Some abnormal ventilation*
- *Lung volumes*
- *Dynamic tests for ventilation*
- *Pleural cavity*
- *Abnormal chest and lung movements*
- *Controlled respiration*
- *Diffusion respiration*
- *Respiratory movements in anesthesia*
- *Compliance, resistance and time constant*
- *Work of breathing*

Respiration involves movement of air in an out of lungs and it is accomplished by the highly sophisticated respiratory apparatus which is mainly controlled by a central mechanism which is finely modulated by various inputs from periphery.

The control mechanism and the peripheral inputs have been discussed in physiology of respiration.

The respiratory apparatus, its structure and the mechanism by which it performs the respiratory functions is known as ***"Mechanics of respiration"***

The knowledge of the mechanics is essential, so that it could be usefully applied during mechanical ventilation.

THE RESPIRATORY APPARATUS

The respiratory apparatus is the combination of various anatomical structures which work in coordination to perform the work of gas exchange between the atmosphere and the organism. It consists of three components. They are,

- The lungs and the airway – (the tracheobronchial tree)
- The thoracic cage – (in which the lungs are housed)
- The muscles of respiration – (which does the work for moving air in and out of lung).

1. The lungs and tracheobronchial tree

- The detailed anatomy has been discussed earlier in "Anatomy of respiratory system" (Chapter 3).

2. Muscles of respiration

Based on their contribution in the process of respiration, the muscles of respiration are classified into three groups. They are,

- Inspiratory muscles
- Expiratory muscles
- Accessory muscles

3. Thoracic cage

- The thoracic cage formed by vertebrae behind, ribs on the side, and the cartilages and sternum in front. This has been discussed earlier in anatomy (in Chapter 3).

MUSCLES OF RESPIRATION

- Diaphragm – (this is the principal muscle of respiration)
- Intercostal muscles
- The abdominal muscles:
  - External oblique
  - Internal oblique
  - Transverse abdominis
  - Rectus abdominis

Among these muscles,

- The diaphragm is the principal muscle of respiration, particularly for inspiration at rest.

- The intercostals are also muscles of inspiration taking part in active respiration.
- The abdominal muscles are basically muscles of expiration, which help in active expiration.

All the muscles of inspiration have *one thing in common; **they are attached to the thoracic cage.*** Similarly, all expiratory muscles have their ***one end attached to the thoracic cage***.

- During quiet breathing, inspiration is produced by contraction of two primary muscle groups, the diaphragm and the intercostals.
- *About 60% of tidal volume is contributed by the diaphragm,* with the remainder being primarily contributed by the external intercostals.
- Since the vital capacity (4–6 liters in an adult) far exceeds the normal tidal volume (500 ml) it is apparent that individuals can survive indefinitely if they lack any one of the systems, provided the other is intact.
- This explains how patients undergoing cesarean section with a T2 level of spinal anesthesia can breathe adequately using their diaphragm alone, while patients with bilateral phrenic nerve block or cervical epidural anesthesia can breathe using only their intercostals.
- Ideally, intercostal and diaphragmatic movements are coordinated; otherwise, the descending diaphragm can "suck in" the chest wall, resulting in paradoxical movements of the chest wall and the abdomen with decreased effective gas exchange.
- Even during quiet breathing, expiration may not be entirely passive; there is evidence that contraction of abdominal muscles may promote expiration.

Diaphragm

Diaphragm means a *separator.*

This has ***a central tendon*** arched on both sides called as ***cupola.*** Muscles radiate on all sides from this. The muscle fibers have three portions,

1. ***Spinal or crural portion:*** arises from the upper ***3 or 4 lumbar vertebrae*** and from the ***arcuate ligament***. Inserted into the posterior margin of the central tendon.
2. ***Costal portion:*** arises by a series of digitations from the ***inner surface of the lower six ribs and cartilages.***
3. A small contribution from ensiform process or xiphoid process.

The central tendon domes up into the thoracic cage, because of the increased intra abdominal pressure and the negative pressure in the thoracic cage.

Nerve Supply

- Phrenic nerve – 90% Motor and 10% sensory and autonomic.
- The two crurae receive motor supply from the 11th and 12th thoracic nerves.
- Peripheral part of diaphragm receives sensory and autonomic from lower six intercostal nerves.

Movements

- In quiet respiration diaphragm moves 1.5 cm vertically downwards.
- May extend up to 6 to 10 cm, in deep breathing.
- Change in posture does not affect this considerably, but obstruction to abdominal wall movements may affect it severely.
- Postoperative propped up position does not do any good in improving the movements.

- Descend of diaphragm by 1 cm causes 350 ml tidal volume. Therefore descend of 1.5 cm causes 500 ml tidal volume.
- In quiet breathing 100% tidal volume is made up by the diaphragm.
- In deep breathing 75% by diaphragm and 25% by rib movement.
- In bilateral phrenic nerve palsy when the diaphragm is paralysed, tidal volume is greatly reduced, still adequate tidal exchange for rest and light activity is well maintained.
- Unilateral phrenic nerve palsy, there is no change in the tidal volume, but 15–20% reduction in the maximum breathing capacity (MBC), and paradoxical movement will be seen.

Intercostal Muscles

External Intercostals

This muscle mostly occupies the posterior part of thorax. It runs obliquely downwards and forwards from the outer and lower border of the upper rib to the upper outer border of the lower rib.

Contraction of this muscle causes the rib pulled closer, but by the virtue of articulation, the rib moves upwards, and outwards. In upper thorax the anteroposterior diameter increases and in lower thorax the transverse diameter increases.

Internal Intercostals

This muscle mostly occupies the anterior part of thorax. It runs downwards and backwards from the floor of costal groove and corresponding costal cartilages to the upper

and inner border of the lower rib. The fibers cross each other. This muscle is supplied by the intercostal nerve. Movements of 5th to 9th intercostals contribute for the ***"Bucket handle movement"*** of the ribs causing increase in transverse diameter of thorax.

Abdominal Muscles (Expiratory Muscles)

They are external oblique, internal oblique, transverse abdominis, and rectus abdominis. These are bilateral muscles.

Movements

- During quiet respiration and up to a ventilation of ***40 L/min (5 times the normal), they are inactive.***
- When there is a further increase in ventilation, abdominal muscles slowly take part in expiration.
- By the time forced contractions are seen, the minute volume is already ***90 liters/min.***
- Earliest movements are seen at the end of expiration.
- The maximum intra abdominal pressure that can be maintained by a conscious person for a few seconds is 110 mm Hg. During electroconvulsive therapy it may rise to 150–200 mm Hg.
- Abdominal muscles do not take part in inspiration, during expiration they maintain the stable intra-abdominal pressure that helps in ascend of diaphragm. The lower ribs are drawn downwards and medially.
- They help in coughing by increasing the intra-abdominal pressure.

Accessory Muscles of Respiration

- *Scalene muscles* – support the apex of lung.
- *Sternomastoid* – contracts during dyspnea.
- *Trapezeus, serratus anterior, latismus dorsi and pectoralis muscle.*

Many of these muscles contract during cough. If, the nerve to latismus dorsi gets injured, during radical mastectomy, the paralysis of that muscle results in ineffective coughing.

NORMAL LUNG MOVEMENTS

Those parts of the lung in direct relation to the mobile and expansile parts of the thoracic cage expand well. Therefore, the peripheral parts of the lung undergo greater degree of expansion than that near the hilum. *There are three areas that are not directly expanded.*

1. Mediastinal surface in contact with pericardium.
2. Dorsal surface in contact with the spinal segments of ribs.
3. Posterior apical surface lying close to the deep cervical fascia of Sibson.

During each inspiration the capacity of the thoracic cage increases in *transverse*, *anteroposterior* and *vertical* diameters.

Apex

- The thoracic inlet, formed by the first two ribs, vertebral column, and manubrium sterni.
- Moves upwards and forwards on inspiration to increase the *anteroposterior diameter* of the chest wall.
- In that way, the a*nterior part of apex or the upper lobe expands directly.*
- After the age of 60 years, the manubrium sterni angle gets ankylosed and this part of lung stops expanding.

Thoracic Cage

- Upper part: from 2nd to 6th ribs.
  - In the upper part, mainly *anteroposterior diameter increases.*

- Lower part: From 7th to 10th ribs.
  - In the lower part, mainly *transverse diameter increases.*

The Diaphragm

- As this is principal muscle of inspiration, in normal breathing, the whole ventilation is done by this muscle. In maximal inspiratory effort, 60% is done by this muscle.
- Therefore, lower parts of the lung, "the bases" undergo the ***greatest degree of movements.***
- ***In supine position*** abdominal muscles relax, the intestines push the diaphragm up and so the diaphragm has the highest potential for contraction.
- In erect posture, the intestines fall down, so the diaphragm contracts inefficiently.

Other Factors

- Vertical movements can be caused by flexion or extension of vertebral column.

NORMAL MOVEMENTS OF RESPIRATION

Normal movement of gases is based on two factors which are known as gradients,

- One is from an area of *higher concentration* it flows to an area of *lower concentration.*
- The other is from an area of *higher pressure* it flows to an area of *lower pressure.*

The basic principle by which the air enters the lung during normal respiration is related to various pressures. They are,

- *Atmospheric pressure*
- *Intrapulmonary (alveolar) pressure*

- *Intrapleural pressure*
- *Transpulmonary pressure*

The process of a respiratory cycle is as follows,

- At rest, the pressure within the alveoli is atmospheric. (0 cm H_2O)

Inspiration

- When a spontaneous inspiration is initiated, the contraction of the diaphragm and intercostals muscles occur, thus putting the muscular work for breathing.
- Thus inspiration is an active process requiring expenditure of certain amount of energy.
- The contraction of the inspiratory muscles enlarges the thoracic cavity.
- The lungs expand because they are pulled out wards along with the thoracic wall.
- The lungs move with the chest wall because of the surface tension created by the small amount of fluid present in between the visceral pleura and the parietal pleura.
- *This expansion of lung increases the volume of the lung and the alveolar pressure drops a little creating a negative pressure* (subatmospheric) (Fig. 6.1).
- This *gradient* of pressure between *the atmosphere and the alveoli* causes the flow of air into the alveoli through the tracheobronchial tree which is open to the atmosphere through glottis and upper airway (Fig. 6.1).
- The air need not be sucked into the lung; air simply moves from the area of high pressure to the area of low pressure to equalize the pressure.
- The intrapleural pressure is normally negative because of the inward pull caused by the elastic recoil of the lung, becomes more negative during the inspiration.

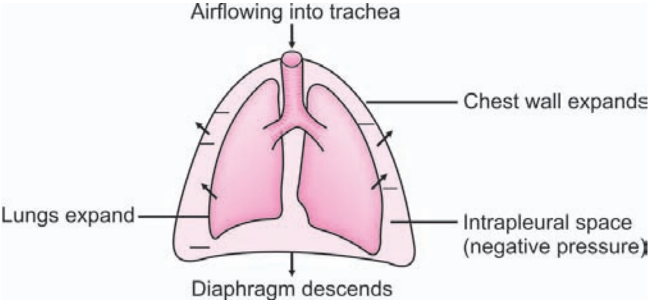


Fig. 6.1: Thoracic cage and lungs in normal inspiration
❖ The expansion of thoracic cage is followed by the lungs.
❖ Intrapleural negative pressure increases.

- Inspiration will continue till the intra-alveolar pressure becomes atmospheric. (Till the pressure gets equalized)

Expiration

- Expiration is a passive process that occurs because of the elastic recoil of the lung.
- When the contraction of the inspiratory muscles ceases, the thoracic cage and the lungs will recoil to their original size. Intrapleural pressure becomes less negative.
- Intra-alveolar pressure becomes slightly positive during expiration because of the squeezing effect on the lungs and once the expiration ends, again it will become atmospheric (Figs 6.2 and 6.3).

The increased negativity in the intrapleural pressure during inspiration has two important roles,

- Allowing the flow of air into the lungs
- Promoting venous return to the right side of the heart (preload) by expanding the great veins.

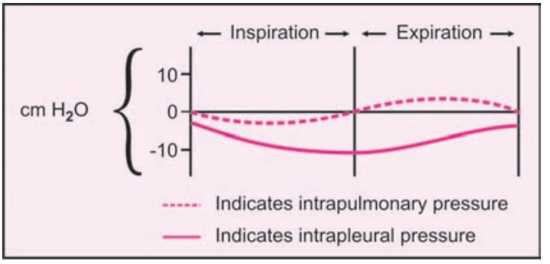


Fig. 6.2: Normal respiration.

- ❖ Changes of pressures in alveoli and in intrapleural space.
- ❖ Note that during inspiration intrapulmonary pressure becomes negative by 1 to 2 cm H_2O and becomes minimally positive (1 to 2 cm H_2O) during expiration.
- ❖ The resting value of intrapleural pressure is –5 cm H_2O at the start of inspiration, slowly increases to –10 cm H_2O at the end of inspiration and slowly returns to the original value of –5 cm H_2O at the end of expiration.

  Now this can be compared to the changes in pressure that occurs during positive pressure ventilation with the help of the figures below.

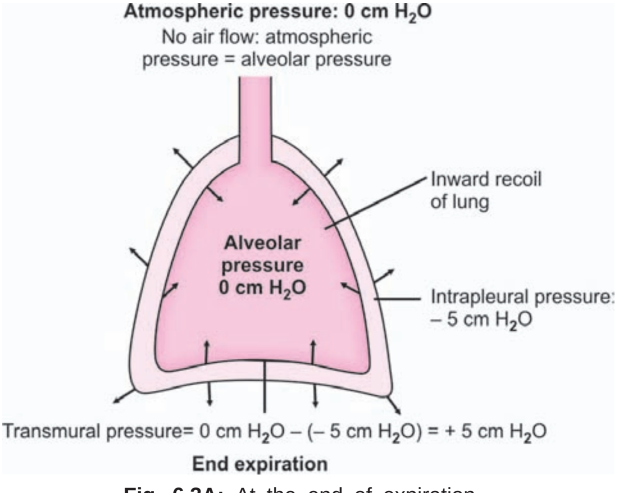


Fig. 6.3A: At the end of expiration

- ❖ **A**. Intra-alveolar pressure and atmospheric pressures are equal (0 cm H_2O).

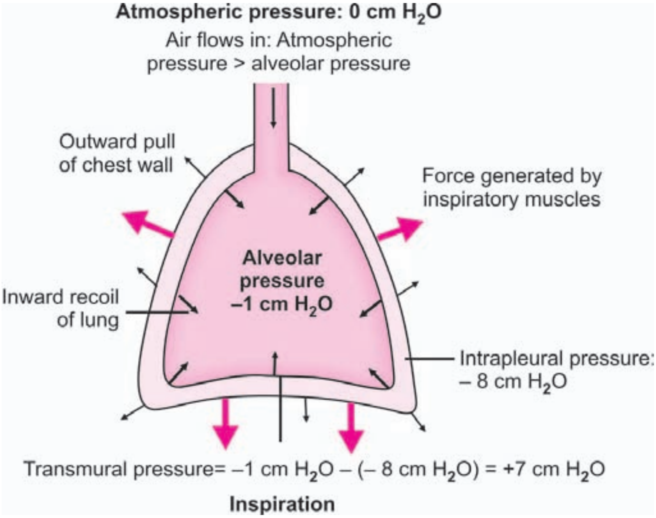


Fig. 6.3B: During inspiration

❖ **B.** Pressure in the alveoli becoming negative (–1 cm H_2O) and the atmospheric air is drawn into the lungs.

All the pressures become positive during positive pressure inspiration (mechanical ventilation). Airway pressure, intra-alveolar pressure and even the intrapleural pressure which always is negative in normal respiration is made positive (Fig. 6.4).

Application of positive pressure within the thorax in mechanical ventilation affects the distribution of gases and may cause hemodynamic embarrassment, primarily through reduction in right heart preload (Fig. 6.5).

SOME ABNORMALITIES OF VENTILATION

- Normal respiration is a rhythmic spontaneous activity; one is quite unaware of it happening and is controlled by the respiratory center.

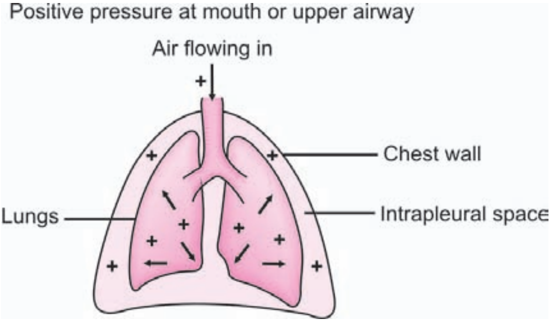


Fig. 6.4: Positive pressure inspiration

- ❖ Note that all the pressures, airway pressure, alveolar pressure and the intrapleural pressure have become positive.

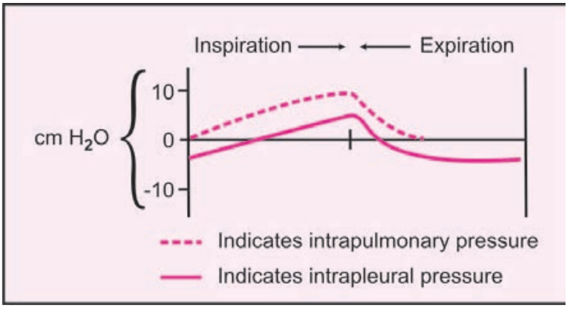


Fig. 6.5: Pressure waves during positive pressure respiration

- ❖ Note that intra-alveolar pressure which is normally atmospheric pressure becomes positive at the start of inspiration and reaches the peak (about +10 cm H_2O) at the end of inspiration and drops back to atmospheric during expiration.
- ❖ The intrapleural pressure which is about – 5 cm H_2O at the start of inspiration becomes positive and reaches the peak of about + 5 cm H_2O at the end of inspiration and drops back to the original value of – 5 cm H_2O during the expiration.

- *If an individual starts having the awareness of his own respiration, it is not normal.*
- It may be physiological as during exercise there will be increased ventilation in response to the increased production of carbon dioxide, and it is usually a pleasurable feeling.

There are some conditions and terminology used to note some type of abnormal ventilation.

Tachypnea

It is an increase in the rate of respiration. Work of breathing is minimal. Usually it is on psychological reasons.

Hyperpnea

Increase in ventilation in relation to CO_2 production. Therefore, the $PaCO_2$ is normal, e.g. commonly occurs after exercise, in hyperthyroidism where general body metabolism is increased. There may be increase of 50–100% in ventilation.

Hyperventilation

Increase in ventilation out of proportion to CO_2 production. e.g. metabolic acidosis as in renal failure, and in diabetic ketoacidosis.

Hypoventilation

Decrease in ventilation. Arterial PCO_2 is increased, e.g. Overdose of opioids may cause this by depressing the respiratory center. It is seen in general anesthesia in spontaneous ventilation to some degree.

Dyspnea

It is a subjective sensation which is difficult to define. It is ***inappropriate result*** (e.g. volume of inspiration) for a ***given muscular effort*** or greater effort than expected is required to produce a given volume of ventilation. In other words the work of breathing is increased and that causes unpleasantness or distress to the patient.

It may be defined as,

"Unpleasant awareness of ones own breathing, due to the increased work of breathing which is felt as a distress"

It is not ***'breathlessness'.*** This term is refers to a physiological state after exercise and is a pleasurable feeling.

LUNG VOLUMES

- The total air in the lungs is divided into sub divisions called **"Volumes".**
- When two or more volumes are added, they are called as **"Capacities"** (Fig. 6.6).
- Air within the lung (some volumes and capacities) can be measured with an instrument known as Spirometer.

It is important to recollect the various volumes and capacities related to the lung and ventilation. The measurement of the lung volumes is useful because many pathophysiologic states alter the lung volumes. The effect of therapy instituted may improve the volumes which can be assessed periodically (Fig. 6.7).

There are four volumes and four capacities. All these can be calculated ***based on the tidal volume***. The normal range of tidal volume is 5 to 8 ml/kg body weight.

The Four Volumes:

• Tidal volume:	500 ml.
• Inspiratory reserve volume:	1500 ml.
• Expiratory reserve volume:	1000 ml.
• Residual volume:	1500 ml.

The Four Capacities:

• Vital capacity:	3000 ml.
• Total lung capacity:	4500 ml.
• Inspiratory capacity:	2000 ml.
• Functional residual capacity:	2500 ml.

Definitions and Clinical Significance

Tidal Volume (V_T)

It is the volume of gas moved into or out of the lung in a single normal inspiration or expiration.

The average volume is 500 ml, or 5 to 8 ml/kg.

It represents the volume reaching the alveoli, about 350 ml, (*That is the air which carries oxygen to the alveoli and*

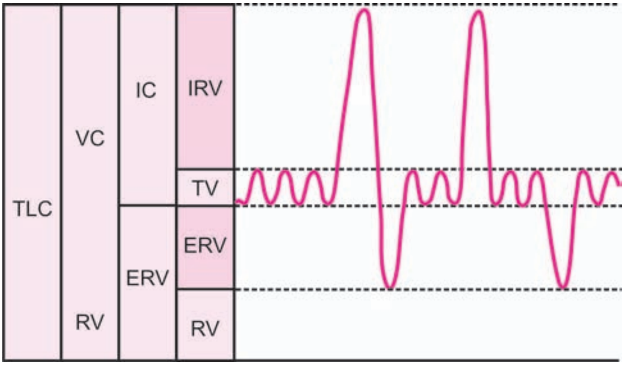


Fig. 6.6: Various volumes and their compositions making the capacities

❖ Note that the combination of volumes make capacities.

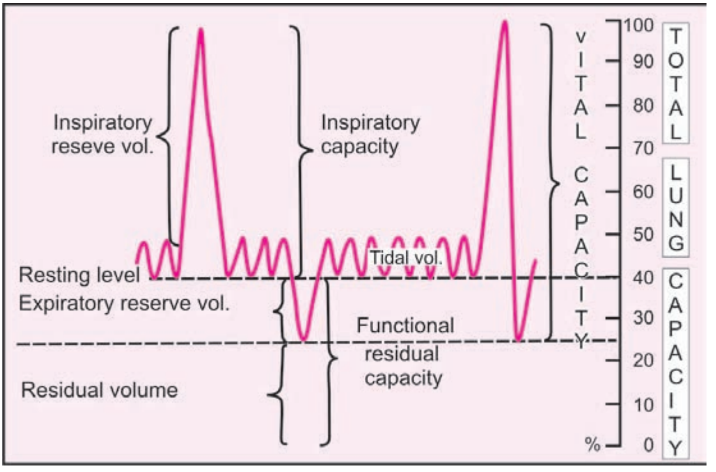


Fig. 6.7: The four volumes and four capacities

❖ Note that the volumes and capacities by percentage of total lung capacity.

sends out the carbon dioxide from the alveoli; the alveolar ventilation.) and the volume in the conducting airways, (*which does not take part in gaseous exchange*), known as anatomical dead space air, is about 150 ml or 2 ml/kg.

Hence it is essential that tidal volume is always maintained within the normal value, whether the patient breathes spontaneously, or being manually ventilated. If there is any reduction, the patient may suffer hypoxia and hypercarbia may also be caused.

"It is always better to err on the side of hyperventilation than on the side of hypoventilation."

This famous statement is quoted here only to indicate that the damages caused by hypoventilation can be devastating where as the problems related to hyperventilation is relatively mild. It is absolutely essential to maintain

normal ventilation at all time unless there are special indications to hyperventilate as in the management of cerebral edema.

Another important aspect about tidal volume is the ***"dead space".*** As described earlier, this is the portion of the tidal volume that does not take part in gaseous exchange, not directly coming in contact with the part of lung where diffusion occurs. This is usually one third of the tidal volume, *anatomical dead space.*

When a breathing system is employed for delivering the gases to the patient's respiratory tract, this inevitably adds a variable space to this anatomical dead space. This is known as ***"apparatus dead space"*** (Fig. 6.8).

It is essential to see that the apparatus dead space is reduced to as minimum as possible otherwise it will

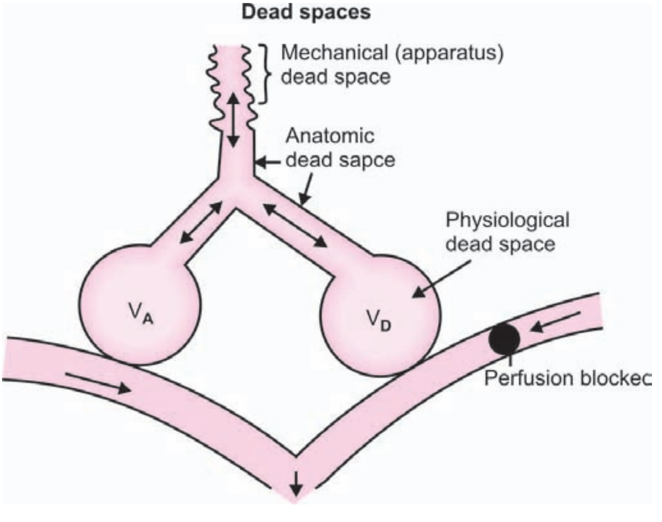


Fig. 6.8: Anatomical dead space, apparatus dead space

❖ Note, ventilation to the alveolus not perfused as shown in the figure is wasted and adds to dead space.

encroach on the alveolar ventilation (that part which takes part in gaseous exchange) and cause hypoxia.

- Intubation reduces the anatomical dead space by about 70 ml.
- An anesthetic face mask may add about 60 to 70 ml of apparatus dead space.

Inspiratory Reserve Volume (IRV)

It is the volume of air that can be inspired at the end of a normal tidal inspiration.

- It is appropriately known as "reserve" volume as this volume is called into action when there is need for increased tidal breathing as in exercise.
- It is approximately three times the tidal volume (about 1500 ml).

Expiratory Reserve Volume (ERV)

This is the maximal volume that can be exhaled after a normal expiration.

- It is approximately about two times the tidal volume. (1000 ml)

Residual Volume (RV)

This is the volume of air remaining in the lungs after a maximal expiration.

- It is the volume of air that is present in the lung after a maximal expiration following maximum inspiration.
- This is the amount of air that cannot be expelled out of lung at any time.
- This volume cannot be measured by spirometry. It can be measured indirectly by using the helium dilution test or body plethysmography to determine the Functional

residual capacity (FRC). The formula **RV = FRC – ERV** is used to determine **RV.**

- This volume is approximately three times the tidal volume, i.e. 1500 ml .
- When the residual volume is increased it means that the lungs are larger than normal and cannot empty the air adequately. It means that there is *"Air trapping".*

This condition is usually found in patients with chronic bronchitis or asthma and in chronic emphysema. Because if the inadequate emptying of the lungs there can be retention of secretions in these patients in the post operative period leading onto lung complications. These patients may require support with drugs and chest physiotherapy to prevent such complications.

Inspiratory Capacity (IC)

$$IC = IRV + V_T$$

This is the maximum volume of air that can be inspired after a normal expiration.

- Measurement of IC as a determinant of maximal tidal volume capability is more useful and accurate than IRV. That is because while measuring IRV deciding where the inspiration ends is very difficult.

Functional Residual Capacity (FRC)

$$FRC = ERV + RV$$

This is the volume of air that remains in the lungs at the end of a normal expiration.

- The clinical significance is that this is the volume of air *where the gas exchange is constantly taking place.*
- *The tidal volume (V_T) can be thought of as a dilutional volume.*

- The tidal breaths bring in fresh gas to mix with the volume already present in the lungs, the FRC where steady state gas exchange is occurring.
- In many pathological conditions such as atelectasis, secretions, or fluid collections in the lungs, or pleural effusion, the FRC is reduced and thus the gas exchange is affected.
- Direct measurement of the FRC is not possible with spirometry.

Clinical importance:
This the expiratory reserve volume and the residual volume put together. *This is the residual air to which the tidal volume gets mixed to reach the alveoli for diffusion.* Actually this air is the ready source of oxygen to get into the blood when the patient is apnoeic. Therefore, when there is an expected delay or difficulty in intubation it is advisable to denitrogenise the lung by making the patient breathe 100% oxygen through the mask for 3 full minutes. This removes all the nitrogen from the lungs and lung is filled with 100% oxygen so also the FRC is full of oxygen. This patient will not become hypoxic even when he is apnoeic for nearly 6 minutes. However, as carbon dioxide removal requires ventilation, there will be hypercarbia.

The FRC is less in small children and so they quickly develop hypoxia if not ventilated even for a very short time. Therefore, they have to be oxygenated well before intubation and delay in intubation must be avoided.

- In adults FRC is 34 ml/kg. and in small infants it is 30 ml/kg.

Vital Capacity (VC)

$$VC = IRV + V_T + ERV$$

• Tidal volume	500 ml.
• Inspiratory reserve volume	1500 ml.
• Expiratory reserve volume	1000 ml.
• Vital Capacity	4000 ml.

This is the volume of air that is exhaled after the deepest possible inspiration by forceful expiration.

- This volume can be measured with a hand held spirometer at the bedside in a cooperative patient.
- The clinical significance of this is*; if a patient has his vital capacity reduced to 3 times the tidal volume,* artificial support may be needed for clearing his secretions.
- It is clinically significant and useful that it tells us the patient's maximal ventilatory capability.
- VC is often measured and trended as an indicator of the patient's ability to be weaned from mechanical ventilation.

Vital capacity can be reduced in

- Alteration in muscle power. As in myasthenia gravis or by use of muscle relaxants, etc.
- Pulmonary diseases like chronic bronchitis, fibrosis, and lobar collapse, etc.
- Space occupying lesions in the chest.
- Abdominal tumors, tense severe ascitis, etc.
- *Gravid uterus does not decrease the vital capacity*; in fact it increases by 10% above normal because of anatomical changes that in lower thorax occur during pregnancy.
- Abdominal pain.
  - Upper abdominal: 70 to 75% reduction of VC.
  - Lower abdominal: 50% reduction of VC.
- Abdominal splinting as tight bandages.
- Positions: Various positions have different effect in modifying the vital capacity. Of that maximum reduction is seen in,

- Trendelenberg: 14.5% reduction.
- Lithotomy: 18% reduction.

Total Lung Capacity (TLC)

$$TLC = IC + FRC$$

This is the maximal volume of air in the lungs after a maximal inspiration.

- It is a total sum of all the lung volumes.

Closing Volume (CV)

- When the lung empties the air, the lung becomes smaller and the small airways begin to close so that no further expulsion of gases is possible. The lung volume at this point is called ***"Closing volume".***
- This volume cannot be measured with a spirometer.
- This is expressed as a percentage of the VC and is normally 10% but may increase with age and disease processes that lead to loss of lung elasticity.
- When the closing volume is greater than the FRC, then arterial hypoxaemia is sure to occur.
- The airways in the basal portions of the lung close sooner than those above because the transpulmonary pressure gradient is less at the base (Fig. 6.9).
- The reason is that intrapleural pressure is less negative in the bases because of the weight of the lungs hanging in the thorax.
- When the lung elasticity or volume decreases, the intrapleural pressure at the base of the lung may actually become positive, compressing the lungs.
- Under these conditions, closing volume (CV) increases and alveolar ventilation decreases.

All these values when estimated, must be interpreted with due caution, as there is a wide margin for normal values even in healthy individuals.

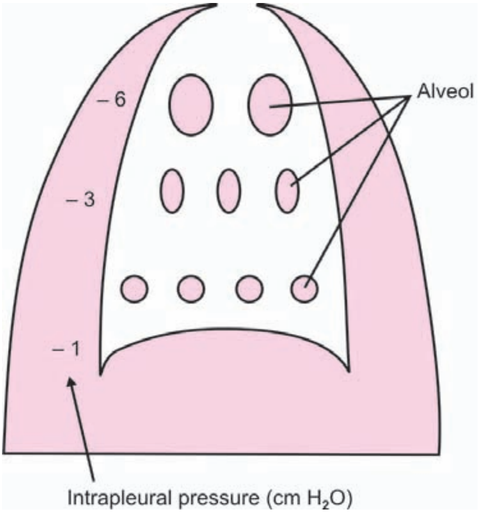


Fig. 6.9: The variation of intrapleural negative pressure between the apex and base

- ❖ Note that the negative pressure is less at the base and more at the apex.
- ❖ Note that *transpulmonary pressure gradient* is lower at the base.
- ❖ Alveoli are more distended at the apex and less at the base.

DYNAMIC TESTS FOR VENTILATION

Maximum Breathing Capacity

It is the maximum volume of air that can be breathed per minute by the individual.

This has to be tested carefully as there may be errors. Secondly it is really tiring to the patient particularly in very ill. Therefore, it can be tested for 15 seconds and calculated for one minute.

MBC gets reduced as age advances, in emphysema, bronchospasm etc. This test may be used to judge the effectiveness of bronchodilator treatment.

Value in 15 second × 4 = must be about 100 to 200 liters. Because of the practical difficulty in educating the patient and the highly variable values, it is not done routinely.

Forced Expiratory Volume (FEV)

It is forced vital capacity.

The patient makes a maximal inspiration and expiration is done as forcefully and rapidly as he can into a spirometer and the total amount of air expelled in a given time is measured. This is a very useful test and easy to do and repeat if needed. The value in the first second is known as **$FEV_{1.0.}$** This volume expressed as percentage of the forced vital capacity. The normal value is from 70 to 83 percent.

In patients with chronic bronchitis, it may be as low as 50 percent.

Peak Expiratory Flow Rate

This is the speed or velocity of air during the peak of expiration and it is mentioned as L/min. Normal value is 450 to 700 liters/min for men and 300 to 500 liters for women.

This is ***an expression of the speed or velocity with which the air flows out of the airway at the peak of expiration when the velocity is at its maximum***. In other words in a normal individual the velocity is anything between 300 to 700 liters/min.

After a maximal inspiration, the patient does the expiration as forcefully as he can, and the maximum flow rate of air is measured. This can be done by the instrument, pneumotachograph, or Wright's peak expiratory flow meter. It may be done for five times and the average is taken.

Clinical Significance

If the value is lower than normal, it indicates mainly obstruction to the airway. In obstructive disease like asthma and chronic emphysema the value is low.

Lower values of $FEV_{1.0}$ and PEFR usually indicate a higher resistance to air flow within the conducting airway. e.g. diffuse airway obstruction.

Some patients have a reduced vital capacity, although their PEFR and $FEV_{1.0}$ values are within normal limits. This type of ventilatory defect is described as **"restrictive"** type. This may occur in alteration in muscle power.

PLEURAL CAVITY

This is a potential space between the thoracic cage and the lungs between the two layers the pleura (parietal and visceral layers) with a thin film of lymph to lubricate it. Because of the cohesive force, *the pressure in this cavity is always negative*. This negative pressure is increased by the elastic recoil of the lungs. Though in normal conditions the pressure is always negative, when the pleural cavity is open the pressure becomes atmospheric and because of the elastic recoil, the lung collapses.

The elastic recoil of the lung is due to,

1. The elastic tissues present in the lung and interstitial tissues.
2. *"Geodesic"* arrangement of muscle fibers on the bronchial tree so that when they contract the length as well as the diameter gets reduced (see Chapter 3).
3. The surface tension of the fluid lining the alveoli.

The normal pressures in the pleural cavity

- – 5.6 mm Hg in inspiration (– 10 cm H_2O)
- – 2.6 mm Hg in expiration (– 5 cm H_2O)

- With the glottis closed, – 40 mm pressure can be reached.
- With glottis closed forced expiration can make the pressure positive to + 50 mm Hg.

This change of pressure in pleural cavity has little bearing on the thoracic cage. *However, these cause effect on heart and thin walled vessels. Venous return either increase or decrease depending upon the change.*

Intrapleural Pressure and Pulmonary Collapse

When collapse occurs in any part of lung, the surrounding tissues will be pulled in, the negative pressure increases. The diaphragm being the mobile structure, it will be pulled and rise up.

PNEUMOTHORAX

When air enters in between the two layers of pleura (the parietal layer and the visceral layer) the negative pressure is no longer present, the layers move away and the lung collapse due to recoil. The presence of air in the pleural cavity is known as ***"pneumothorax".*** This can be due to various causes and be of various types. Whatever be the cause and type any pneumothorax can cause variable degree of haemodynamic disturbances as well as disturbances in lung functions, some of them may be fatal.

Any type of pneumothorax may occur in a critically ill patient with pulmonary problems and hence identifying them promptly and managing them appropriately is essential.

Types of Pneumothorax

- Open pneumothorax: Bronchopleural fistula or open wound of chest wall.

- Closed pneumothorax: Pleural cavity has no communication to atmosphere.
- Tension pneumothorax: Ball valve type communication. It is potentially a fatal condition. It may mimic severe bronchospasm during anesthesia and may be fatal if not treated immediately.
- Artificial pneumothorax: Pneumothorax was induced deliberately as a treatment for pulmonary tuberculosis in olden days (Now it has no relevance).

Open Pneumothorax

The pleural cavity is in communication to the atmosphere, most commonly through a opening in the thoracic wall.
- The size of the opening in relation to the diameter of trachea is important.
- Larger opening; the patient can quietly breath, though there is no negative pressure in pleural cavity and the lung is partially collapsed.
- If it is an opening in the chest wall, IPPR will correct the problem and improve the condition.
- If it is a broncho-pleural fistula then IPPV may cause "Tension pneumothorax".

Closed Pneumothorax

It may occur after thoracotomy closure, or due to rupture of an emphysematous bulla.

The gases may be removed by:
1. Absorption: The visceral pleura is permeable for gases. Therefore, it is slowly removed.
2. Aspiration: Can be done, but not commonly employed.
3. Water seal: Works as a drainage. Works as a unidirectional valve to empty the gases.

4. Suction through the water seal: This also is not advisable, as it may make the leak persistent which otherwise gets sealed.

Tension Pneumothorax

When the air enters the pleural cavity continuously, commonly through a one way valve like opening, and is not able to leave it, tension builds up in the pleural cavity and is known as "Tension pneumothorax".

- When positive pressure ventilation is employed, an emphysematous bullous may rupture or through a small broncho pleural fistula air may leak into the pleural cavity.
- A normal lung will not rupture with ordinary inflation pressures, it requires very high inflation pressure to rupture it.
- A *contused lung after multiple injuries* may rupture with IPPR. Hence care is taken to avoid unduly high inflation pressure.
- In *pulmonary tuberculosis rupture of bullous is possible* spontaneously during cough.
- During anesthesia the characteristic feature will be sudden loss of resistance followed by quick increase in resistance making the reservoir bag stony hard.
- Patient's condition rapidly deteriorates with cyanosis, hypotension, and cardiac arrest.
- This condition mimics severe bronchospasm with absolutely no improvement for treatment.
- Percussion of chest reveals hyper resonance. A wide bore needle introduced in the second intercostals space will release the gas under tension and the condition improves quickly. This procedure only will save the life of the patient.

In conscious patient sudden onset of cyanosis, hypotension and dyspnea may occur. Here again it may be mistaken for bronchospasm and percussion of the chest will differentiate it from bronchospasm.

Tension pneumothorax causes rapid, progressive shift of mediastinum to the opposite side resulting in severe hemodynamic effects due to the kinking of great vessels and stopping venous return. Tracheobronchial tree is also completely compressed and occluded that no air enters the alveoli.

ABNORMAL CHEST AND LUNG MOVEMENTS

When the stability of the thoracic cage is destroyed either by trauma or by surgical intervention, then abnormal movements of lung and chest wall occur. They may be:

- Paradoxical respiration.
- Pendelluft.
- Mediastinal flap or flutter.
- Flail chest.

Paradoxical Respiration

In crush injuries of chest wall or deliberate surgical removal of a part of thoracic cage as in thoracoplasty, the affected part of chest wall collapses inwards. *On inspiration the unaffected side will expand in the normal fashion but the injured part will be sucked in. On expiration the reverse will take place.* Padding affected side may help in preventing the abnormal movement. This type of paradoxical respiration will be seen only when the patient breathes spontaneously and is abolished by controlled ventilation.

Pendelluft

This is the pendulum like movement of air from one lung to the other in the presence of open pneumothorax in a patient breathing

spontaneously. The physiological result of this pendulum like flow of air is that, the alveolar carbon dioxide tension rises because of rebreathing within the two lungs.

If the opening in the chest wall is small and the patient is breathing quietly, the degree of Pendelluft will be less.

Mediastinal Flap

In man, the mediastinum can move freely with different phases of respiration. It remains however in the middle of the thorax because the negative pressure in the pleural cavities balances each other. In open pneumothorax, because of the negative pressure in the normal side, mediastinal shift occurs to that side, leading to compression of great vessels and atrium. During inspiration the negative pressure increases further and pulls the mediastinum more. During expiration it goes back to its original position.

During quiet breathing even with a large hole in the chest wall it may not be seen but if the ventilation increases the flap also increases. In lateral position it becomes still worse. The normal lung is poorly ventilated. The great vessels are compressed and venous filling to the heart is reduced and cardiac output falls.

Paradoxical respiration, pendelluft, and mediastinal flap are all can be managed by controlling the respiration by mechanical ventilation (IPPR).

CONTROLLED RESPIRATION

Controlled respiration is the scientific basis of mechanical ventilation and actually it is the forerunner of mechanical ventilation.

Taking over the rhythmic respiration manually is known as controlled respiration. It can be initiated in three different ways:

- Paralysis of muscles of respiration.
- Depression of the respiratory center.
- Removing the carbon dioxide stimulus to the respiratory center.

In practice all the three can be used, but in modern days the use of muscle relaxants is commonly employed as the main feature and the other two may contribute for modulating it.

During surgical anesthesia, one of the advantages of controlled ventilation is that the anesthetist can help the surgeon during some delicate, intricate steps of surgery by appropriately reducing the ventilatory movement or temporarily stopping it.

One of the disadvantages is that there can be inadvertent hypoventilation. Though it is commonly said *"it is better to err on the side of hyperventilation"*, this statement holds good only for positive pressure ventilation during anesthesia and not for therapeutic ventilation where the basic intention itself is to maintain normal blood gas levels.

DIFFUSION RESPIRATION

In normal person the interchange of gases at the alveolar capillary membrane depends upon physical diffusion, *while at tidal volumes smaller than 1 liter, the movement of gas from the alveolar duct to the alveoli is almost entirely by diffusion.* Some movement is caused by the contraction of heart and by the ejection of blood from it leading to pulsatile flow in the pulmonary vessels. This causes movement of gases from the distal bronchial tree into the alveoli even if the patient is paralysed and apneic. This process is known as *diffusion respiration.*

If the nitrogen from the lung is removed and it is filled with 100% oxygen by allowing the patient to breath 100%

oxygen for full three minutes, then by this diffusion respiration the oxygenation can be maintained by uptake oxygen in the blood for more than 6 minutes. However, carbon dioxide is not removed from the blood so the alveolar carbon dioxide level equals that of mixed venous blood, i.e. 46 mm Hg.

Diffusion respiration is commonly used in bronchoscopy as "apneic oxygenation" in adults and not in children for safety reasons.

RESPIRATORY MOVEMENT IN ANESTHESIA

Though this part of discussion has no direct relevance to mechanical ventilation but its significance is only to understand how *progressive paralysis of various respiratory muscles is caused, how it depresses the ventilation during anesthesia and its clinical correlation to our subject of mechanical ventilation.* (It is interesting to correlate it with neurological defects for which mechanical ventilation is instituted).

When ether is used for anesthesia as a sole agent, increasing concentrations in the blood are achieved only gradually, so that the consequent effects on respiration are produced ***as a relatively slow and orderly process,*** and are classical of the progressively increasing depth of anesthesia. The signs of progressively increasing depth of anesthesia are well described by Guedel in 1920. ***The characteristic feature is the progressive depression of respiration.***

- The movement of the thoracic cage on inspiration passes steadily from one ***of expansion*** during light anesthesia to one ***of retraction*** in the late stage of anesthesia.
- This transition occurs gradually and must be related to the paralysis of various groups of muscles.

- Thus in light anesthesia the abdominal muscles probably act as synergists and exert an isometric pull on the lower costal margin.
- The chest and abdominal wall rise and fall in unison. (Stage III Plane 1 and 2.)
- As the depth of anesthesia increases, the synergistic muscles drop out one by one.
- In the 2nd plane (Stage III, Plane 2), the respiratory rate, rhythm, and depth are similar to those found in first plane.
- When the plane 3 (Stage III, Plane 3) is reached, a characteristic expiratory pause may occur together with an absence of movement in the upper part of the thoracic cage.
- Now the abdominal element of respiration becomes more marked and the abdomen begins to protrude just before the lower part of the thoracic cage moves outward. In fact the thoracic movements are starting to lag behind those of abdomen.
- When the fourth plane (Stage III, Plane 4) is reached, the inspiratory phase is marked by a quick jerky protrusion of the abdominal wall as the diaphragm descends, followed immediately by a similar jerky retraction of the chest wall.
- This often called as ***"Diaphragmatic respiration"*** and is characterized by paradoxical movement of the abdomen and thoracic cage.
- At this point almost all the muscles of respiration, except the diaphragm, have ceased to function. ***This is a stage of over dosage***. The descend of diaphragm causes protrusion of abdomen, *the negative pressure in thoracic cage draws the thoracic cage inwards.* Expiration is a slow and prolonged movement and is followed by a pause.

- In the fourth stage (Stage IV) the expiratory pause grows longer, inspiration becomes more jerky and irregular, until finally respiratory arrest occurs. This is the stage of bulbar paralysis.

Tracheal Tug

In deep anesthesia, Stage III Plane 3 or 4, inspiration is associated with a tracheal tug. This is a sharp downward movement of the trachea on inspiration. In deep anesthesia when the respiratory muscles get paralysed, the costal fibers of diaphragm contract inefficiently leaving the crural fibers contract. This sharp contraction is transmitted to the whole mediastinum that pulls the root of the lung and trachea downwards.

This is because, the stabilizing muscles of larynx, mylohyoid, stylohyoid, styloglossus and posterior belly of digastric, all of which are the elevators of the larynx are paralysed.

The clinical significance of this tracheal tug in modern anesthesia is that, this condition frequently occurs in patients with:

- Partial curarisation.
- Presence of airway obstruction (even a small spec of thick viscid sputum may be sufficient).
- In shocked states.

Compliance

Compliance literally means ***yielding.***

In respiration, the term compliance is used to refer the ***distensibility*** of the respiratory tissues. *The compliance is the measurement of the lung's distensibility*. When it is easy to inflate the lungs the compliance is said be good.

The elasticity of the lung tissue is due to the presence of elastic and collagen fibers in the interstitial plane. The presence of the interstitial elastic network gives the essential support for the airways, alveoli, and capillaries. Only when, in some diseases these fibers are reduced, the condition is called as "Stiff Lung".

Elastic recoil refers to the return of tissue to its resting position after being stretched, as the balloon deflates or a rubber band returns to its size once the stretch applied is removed. Similarly, the lungs have very strong elastic recoil that makes them want to continually return to their resting state. However, the negative pressure in the intrapleural space is strong enough to oppose the elastic recoil of the lungs. If there is any disruption to it, the lungs collapse immediately.

Distensibility refers to the stretchability of the lung, that is, the relative ease with which the lung can be inflated with the given force.

Compliance is defined as *"the volume change per unit pressure change"*.

- Here the ***volume*** is in ***ml of air*** and the ***pressure*** is in ***cm H_2O.***
- Normal lung compliance is ***200 ml/cm H_2O.***
- In other words, in the process of blowing air into the lung, if 1 cm H_2O pressure is applied to the airway, it will drive 200 ml of air into the lungs.
- Similarly thoracic cage in which the lungs are situated also offer similar resistance and the compliance (distensibility) is known as *thoracic compliance.*
- Normal value of thoracic compliance is ***200 ml/cm H_2O.***

The lung and thoracic wall function as a single unit and provided the tidal volume is in the normal range, there is a linear relationship between the volume change and the pressure that produced it.

- The compliance of the lungs and the thoracic cage together is known as ***Total lung compliance.***
- The normal value of total lung compliance is ***100 ml/ cm H_2O.***
- This is because the lungs in the thoracic cage, offer double the resistance for inflation and so for the given pressure, only 100 ml of air can be forced into the lungs. This is half the volume of air that can be forced by this force when lung alone is inflated.

Static Compliance (C_{ST})

It is measured when the flow of air has ceased as during breath holding or during apnea under anesthesia.

- Lung compliance: 0.2 liters/cm H_2O.
- Thoracic compliance: 0.2 liters/cm H_2O.
- Total compliance: 0.1 liters/cm H_2O.

Total Lung Compliance: **C_{TL} =** Change in volume/Change in pressure.

Dynamic Compliance (C_{DYN})

It is the volume change of thorax in relation to the pressure change is measured during respiration.

The Clinical Significance

Any increase in elastic resistance, structural resistance, and airway resistance will increase the work of breathing and lead onto dyspnea. Similarly when the compliance of the lungs is reduced as in pulmonary edema, fibrosis, congestion, etc. or when the thoracic compliance is reduced as in morbid obesity, structural deformities like ankylosing spondylitis, kyphoscoliosis, etc. the work of breathing increases to cause difficulty in breathing.

Pathological conditions that affect total compliance generally fall into three categories:

- Diseases affecting lung interstitium
- Diseases of the intrapleural space
- Diseases of the chest wall.

Some pathological states which cause reduction in compliance and leads to "Stiff lung";

- Adult respiratory distress syndrome (ARDS)
- Pneumonia
- Pulmonary edema
- Pulmonary fibrosis
- Pneumothorax
- Hemothorax.

A decrease in compliance affects the ventilation. If the patient is not able to increase the work required to generate the greater muscular effort to overcome the stiffer elastic recoil of the lung, a decrease in the tidal volume (V_T) will result.

The distribution of ventilation will also become uneven and will be preferentially distributed to the areas of better compliance.

When the compliance is poor, one has to search for the cause such as congestive cardiac failure, atelectasis, lobar collapse, pneumothorax, pleural effusion and the suitable therapy for improving the compliance must be instituted.

- For example, in consolidation, therapy to mobilise the secretions like suctioning, chest physiotherapy, postural drainage, coughing etc must be started.
- In congestive cardiac failure, diuretic therapy may be instituted.
- In pleural effusion, pneumothorax, hemothorax, or empyema, measures to evacuate the contents and maintaining it with a chest drainage system has to be done.

Measurement of Compliance

The compliance can be measured in some of the modern microprocessor controlled ventilator. They are capable of measuring respiratory mechanics when the operator gives a few inputs and commands.

Measuring Static Compliance

- *Exhaled tidal volume*, *plateau pressure,* and *PEEP* are necessary to calculate this.
- Exhaled tidal volume is more accurate measure of volume actually in the lungs at the time of pressure measurement was taken so it has to be utilized for calculation.
- Plateau pressure was taken by instituting a 2 second inspiratory pause (inspiratory hold) at the peak of inspiration. This pause creates a situation of no gas flowing into the lungs (Fig. 6.10).
- This is the time when there is no gas flow, the glottis is open, the pressure within the alveoli is obtainable as *plateau pressure.*
- This plateau pressure reflects ***the pressure, due to the elastic recoil forces of the lung tissue alone, on the volume of gases in the lung.*** No pressure resulting from the flow of gases is measured.
- Any artificial pressures placed in the airway such as PEEP must be subtracted from the plateau pressure before calculating the compliance (Fig. 6.11).
- Calculation of static compliance is still possible even if the patient does not have an endotracheal tube. This is done by measuring the pleural pressure, which reflects the alveolar pressure, with the help of an esophageal balloon.

C_{ST} = Exhaled tidal volume/Plateau pressure – PEEP

- Normal value of static compliance is **70 to 100 ml/cm H_2O**. This means, for every 1 cm H_2O pressure applied in the airway there a change of volume of 70 to 100 ml of air.

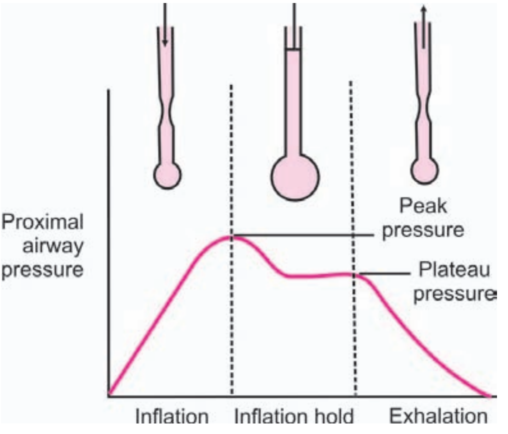


Fig. 6.10: Calculating the plateau pressure

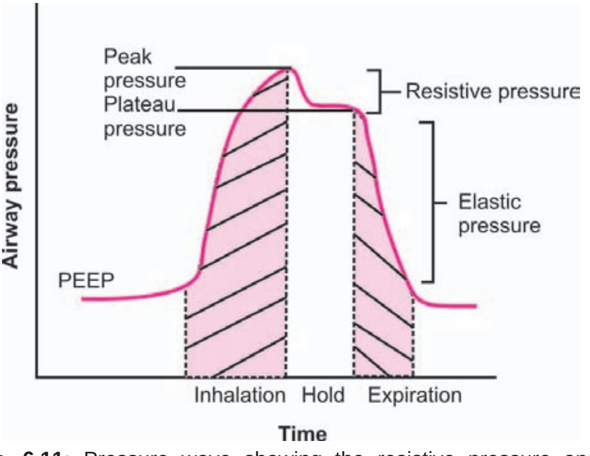


Fig. 6.11: Pressure wave showing the resistive pressure and elastic pressure

❖ In this case the PEEP has to be subtracted from the plateau pressure for calculating static compliance.

Clinical Significance of Static Compliance

- A decrease in the static compliance *tells about abnormalities of lung parenchyma, pleural space, or chest wall.*
- An increase in static compliance is also not indicating something good, but indicates a problem. Increased compliance occurs in disease processes that destroys the elastic tissues of lung as in emphysema. Decreasing elasticity leads to a reduction in transpulmonary pressure, the force that holds the small airways open.
- This may lead to a state, where the small airways decrease in size and may even collapse. This will increase the airway resistance and expiratory flow will decrease.
- Inspiratory work of breathing also will increase when transpulmonary pressure decreases, because greater force is required to achieve sufficient inspiratory flow.

Measuring Dynamic Compliance

- This measurement is done while the gases are moving in the lungs, ***so it measures not only the compliance of the lung tissues but the resistance to gas flow also.***
- This measurement is relatively easier as this does not require an inspiratory hold maneuver.
- But the dynamic compliance is not a pure measurement of lung compliance

$$C_{DYN} = \text{Exhaled tidal volume/Peak inspiratory pressure} - \text{PEEP}$$

- Exhaled tidal volume is measured, as that is the most accurate measurement of volume actually in the lungs at the time pressure measurement was taken.
- This exhaled tidal volume is divided by the pressure in the airway at the peak of inspiration. At this point, gases are still flowing in the lungs, and some pressure

measured will be due to the movement of gases (resistance)

- Any artificial pressures such as PEEP must be subtracted from the pressure measurement when the calculation is done.
- The normal value of dynamic compliance is **50 to 80 ml/H_2O.**
- *Dynamic compliance is always smaller than the static compliance, as the peak airway pressure is always greater than plateau pressure.*

Clinical Significance of Dynamic Compliance

- A decrease in dynamic compliance may *indicate a reduction in lung compliance or an increase in airway resistance.*
- A single value of compliance has no value; a serial measurements and a trend of this variable will give an idea of the improvement following the treatment.

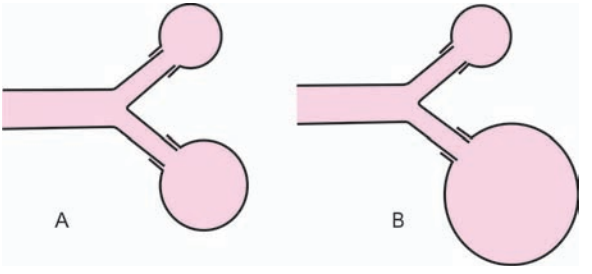
TIME CONSTANT, LUNG COMPLIANCE, AND RESISTANCE

Law of Laplace

"The tangential tension in the wall of a hollow structure is proportional to the internal pressure and to the radius"

If a person blows down the end of a Y piece connection to which are attached two balloons, then one balloon inflates preferentially (Fig. 6.12A).

If, now further air is blown down the connection, one might expect the under inflated balloon will expand. That will not be the case. The already expanded balloon expands even further (Fig. 6.12B).



Figs 6.12 A and B: Illustrating explaining the law of laplace

This phenomenon is due to the law of Laplace. If this law is applied strictly in the case of the lungs, then theoretically a small segment of collapsed lung tissue could never be re-expanded, as any further pressure would only continue to expand the already expanded alveoli.

There is no doubt that alveoli which contain air are easier to expand than those that are completely collapsed. However, prolonged positive pressure will inflate all portions of the lung.

One of the main reasons for this is the presence of surfactant in the alveoli. The remarkable quality of this fluid is that, its surface tension varies directly with the amount of surface area exposed. Thus, *when an alveolus contracts, the tension of the lining secretions automatically decreases.* If it were not for this unusual property, an alveolus once collapsed, would require greater force to re-expand it and persistent collapse would result.

Time Constant

Now, it is easy to understand that when there are differences in the compliance or resistance, there will be uneven distribution of ventilation. This regional difference of ventilation is expressed as ***time constant.***

Time constant (Seconds) = **Resistance × Compliance**

- Different time constants may be present in different regions of lung.
- Areas of lung that have either increased resistance or decreased compliance will have **a longer time constant.**
- Lung units with *longer time constant will need longer inspiratory time to inflate*.
- If the respiratory rate is high, then the lung units with short time constant only will inflate fully.
- The overall results will be regional maldistribution of gases and decreased alveolar ventilation (Fig. 6.13).
- The orientation of the time constant is essential in application of appropriate form of mechanical ventilation using inverse inspiratory to expiratory (I:E) ratios.

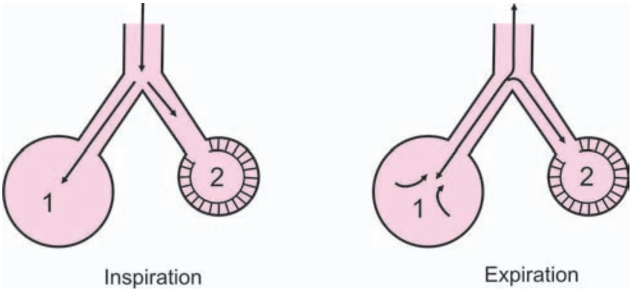


Fig. 6.13: Uneven distribution of gases in lung units with different time constants

- ❖ The alveolus **1** has normal time constant, so the air enters it faster.
- ❖ The alveolus **2** has longer time constant, so air fills it slowly.
- ❖ During inspiration, the unit with normal time constant filled fully well, whereas the alveolus **2** is not filling well.
- ❖ During expiration the alveolus **1** starts expiration while the alveolus **2** is still filling with the gas exhaled from the alveolus 1. This leads to inadequate alveolar ventilation.
- ❖ Application of *inverse ratio ventilation* in this condition, will give a longer time for inspiration that takes care of the underfilling alveoli by filling them slowly and fully.

- Positive pressure ventilation applied on such lungs will result in poor inflation of the alveoli with poor compliance and hyperinflation of alveoli with normal compliance (Fig. 6.14).
- *Inverse I:E ratios* are designed to improve the distribution of gases within the lung, and thus alveolar ventilation, by improving the ventilation *to lung units with longer time constants* (Refer to Mechanics of breathing).

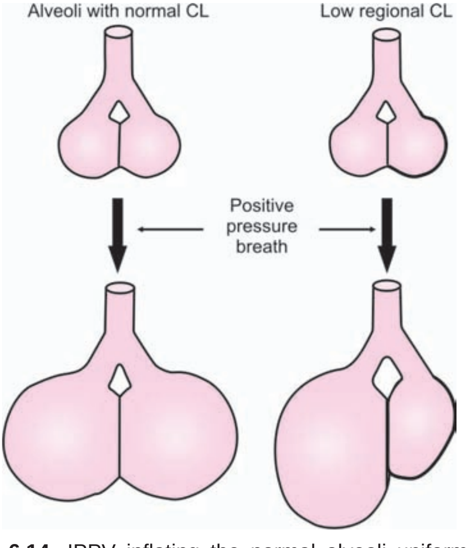


Fig. 6.14: IPPV inflating the normal alveoli uniformly

❖ Normal units are inflated uniformly. Units with poor compliance are poorly inflated whereas the neighboring normal units are hyperinflated.

❖ Regional compliance differences can create regions of poor inflation in diseased units of lung, while grossly over inflating healthier units during positive pressure breathing.

Resistance

Unfortunately the task of respiratory muscle is not simply the inflation of a passive balloon namely the lung. Certain

forces that oppose the inflation of lung have to be overcome to do this.

The resistance is the opposing force to the flow of gases through the airways.

Two types of resistance are encountered in breathing, tissue resistance and the airway resistance.

Tissue resistance caused by various tissues during inspiration and expiration, this constitutes normally 20% of total resistance. This may due to structures of respiratory apparatus and elasticity of the lung.

Airway resistance (R_{AW}) *is the opposing force to the flow of gases caused by the friction between the walls of the airways and the gas molecules, as well as the viscous friction between the gas molecules themselves.*

More easy way of understanding the resistance to breathing is by classifying it into three groups, e**lastic resistance**, **structural resistance** and a**irway resistance**.

Elastic Resistance

- It is defined as the force tending to return the lung to its original size after stretching. This indirectly gives us idea about the compliance of lung.
- If the lung is relatively rigid due to any reason like congestion, fibrosis, etc. then more energy will be needed for inflating it.
- It may be recalled here that, ***compliance of lung*** is a measure of its change in volume per unit pressure change. In other words, the volume of air that can be filled, expressed in ml when 1 cm of water pressure is applied onto the airway (Normally 200 ml).

Structural Resistance

- This comprises of *thoracic wall, diaphragm, and abdominal contents*.

Airway Resistance

- It is dependent on the *length and size of the lumen of the bronchial tree.*
- There may be an extra air resistance added when *an endotracheal tube* is introduced.
- In ***a small child*** who has a small airway it is particularly important, as too small an ***endotracheal tube*** may increase the ***resistance enormously.***
- It is related to the speed of the airflow and it is maximal when the speed is higher.
- The flow of air through the bronchial tree may be ***"laminar"*** or ***"turbulent".***
- The resistance can be lowered in ***laminar flow*** by reducing ***the viscosity***.
- The resistance in ***turbulent flow*** is reduced by lowering ***the density.***

  The resistance to gas flow is measured by using the formula,

$$R_{AW} = \text{Peak pressure} - \text{Plateau pressure/Flow}$$

- Airway resistance is **driving pressure** divided by the flow rate (Fig. 6.11).
- **Driving pressure** is the difference of pressure between the beginning of the circuit, the mouth *(Peak Pressure)* and the end of the circuit, the alveoli *(Plateau Pressure).*
- The flow rate is the speed at which a gas travels or looking at it differently, the volume of gas delivered in a given amount of time.
- Its unit of measure is *liters/minute.*
- The normal value for airway resistance is 0.5 to 3.0 cm H_2O/L per second. Measured at the standard flow rate of 0.5 L/second.

Factors Affecting the Airway Resistance

- ***Length*** of airway
- ***Radius*** of airway
- ***The flow rate.***

Length and Radius of Airway

- When *the length of the airway* is increased, resistance to flow will increase.
- For this reason the length of the ventilator tubing are not too long but standardized.
- A length of endotracheal tube will increase the resistance than a tracheostomy tube.
- *Radius of airway* affects the resistance; *if the size of the airway is doubled the resistance decreases **16** fold.*
- Similarly, if the airway radius is decreased, the resistance to airflow is increased and the flow of air into the lungs is reduced.
- Clinically airway radius can be reduced by the presence of *secretions* in the airway or *bronchiolar spasm*.

The Flow Rate and Flow Pattern

- *Flow rate and flow pattern can affect the resistance.*
- If the flow rate increases, the pressure in the airway increases and so the resistance increases.
- Pressure in the airway rises with the increasing flow rates primarily because of the pattern of flow of gases.
- In **laminar flow**, the molecules of gases in parallel lines to each other, it is streamlined and the pressure is low (Fig. 6.15).
- The molecules in the center move faster than those in the periphery. Those molecules adjacent to the walls will be the slowest. Hence the molecules move with a convex velocity head (Parabolic curve) (Fig. 6.17).

The following figures show the various patterns of the flow of gases in a tube.

Fig. 6.15: Laminar flow

Fig. 6.16: Turbulent flow

Fig. 6.17: Parabolic curve of laminar flow

Fig. 6.18: Laminar flow becomes turbulent in the area of narrowing

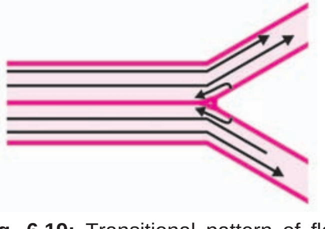


Fig. 6.19: Transitional pattern of flow

- It is created when the laminar flow is disrupted at points where the airways bifurcate.

- This smooth, rounded velocity curve eases into even the smallest airway while creating minimal pressure (Fig. 6.17).
- The **turbulent** flow is disorganized; there are eddies and whorls that create friction between the molecules of gases (Fig. 6.16).
- Turbulent flow, which occurs more commonly when the flow rate is high, causes higher airway resistance.
- Turbulent flow can occur in narrowing in the airway or any kinks in the airways (Fig. 6.18).
- **Transitional flow** is a mixture of laminar and turbulent flow (Fig. 6.19).

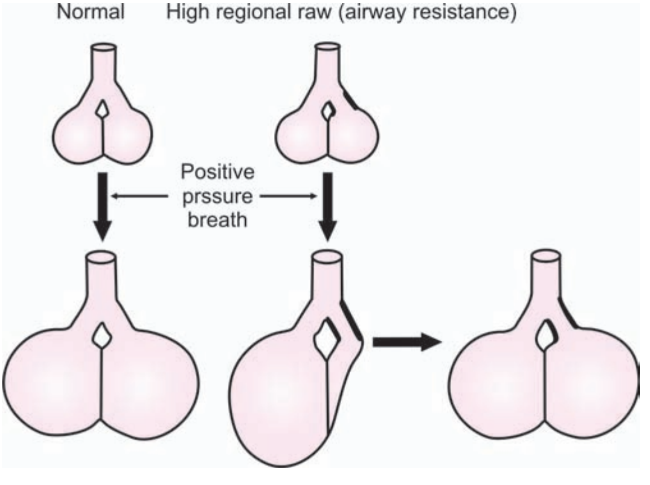


Fig. 6.20: Increased airway resistance (RAW)

- ❖ Regional overdistension and pendelluft effect from heterogeneous distribution due to increased airway resistance.
- ❖ The alveolar units with narrowed airway fill very slowly during inspiration. Whereas there is a transient overdistension of normal alveolus. From this alveolus air flows into the obstructed alveolus by "Pendelluft movement of air".

- When the patient is mechanically ventilated, the *resistance can roughly be assessed by the peak inspiratory pressure (Fig. 6.20).* This can be easily read from the airway pressure gauge manometer.

Patient Factors that Modify Airway Resistance

- Bronchoconstriction, secretions in the airway, bronchial mucosal edema are the common factors.
- The effect of kink in the artificial airway (Endotracheal tube) causing airway obstruction is explained in Figure 6.21.

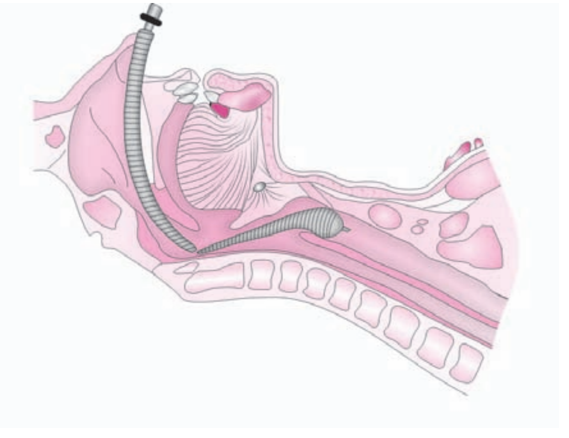


Fig. 6.21: Endotracheal (Nasal) tube getting kinked at the pharyngeal level

Ventilator Circuit Factors that Modify Resistance

- Biting or kinking of the endotracheal tube in bad positioning of the head and neck (Fig. 6.21).
- Kinking of the ventilator tubing between the bed rails and the mattress.

In all these conditions which increase the resistance, gas flow into the lung is reduced if the pressure is not increased.

- Therefore, ultimately, the increase in resistance decreases the patient's tidal volume and the alveolar ventilation.
- When the pressure required to get the air into the lung is increases, *there is uneven ventilation.*
- The areas of lung which has least resistance are well ventilated than the high resistance areas.

WORK OF BREATHING

The mechanics of breathing involves expenditure of energy. The energy is spent by the respiratory muscles for contracting and also for the metabolic work. This work of breathing, though highly significant in clinical terms, is not clinically measurable. (Dupuis,1992; Marini 1990)

However, the work of breathing is the most important factor to be taken into account when considering,

- Initiation of mechanical ventilation
- Choosing the appropriate mode of ventilation
- Altering the ventilator settings
- Weaning a patient from ventilator.

It is defined as ***"All the energy required for ventilating the lungs".***

- Mechanical work is defined as "Force time distance".
- This means that work is done whenever *an applied force causes displacement*.
- In the lungs, *the force is the pressure* and *the volume* (times volume) *is the displacement.*

As discussed earlier, *there are three main forces that cause opposition for ventilation.* They are, **elastic resistance, structural resistance, and airway resistance.** In normal life these forces are kept appropriately at minimum and minimal energy is required for ventilation.

- If any one or more of the opposing forces increase, then more work has to be done for forcing the air into the lungs.
- In general terms, if the lungs are easily inflatable, it is said that the compliance is good and the work of breathing will be normal.
- The work that the respiratory muscles must perform is that which will overcome the *elastic* and *nonelastic* forces: *The compliance* and *the resistance,* respectively.
- When compliance decreases or resistance increases, a greater force is required to move the volume of air into the lung, i.e. The work of breathing (WOB) increases.
- The work of breathing is an important factor to be taken into account when one is considering the initiation of mechanical ventilation, choosing an appropriate mode of ventilation, altering the ventilator settings, weaning a patient from the ventilator.
- The question first asked is, whether the patient has adequate muscle strength to maintain the work of breathing for the compliance and resistance of pulmonary tissue, the work imposed by the pulmonary circuit and the ventilatory demand.
- Physical examination will reveal certain useful clues about the tolerance of the patient for the imposed work.
- If the work of breathing is manageable, the examination will show:
  - A normal respiratory rate
  - Absence of use of the accessory muscles of respiration
  - No abdominal paradox
  - No increase in heart rate
  - No hypertension.
- A clinically meaningful work of breathing is expressed in metabolic terms commonly called *Oxygen cost of breathing.*

- As it is discussed, it is about 4 ml of oxygen/min, of total oxygen consumption per min (it is 1.5%, a maximum of 5% is acceptable).
- In disease processes, where compliance is reduced or resistance is increased, it may be markedly increased as much as 25%. Increased respiratory activity leads to disproportionately high rate of oxygen consumption.

Respiratory Work and Diseases

- It is understood from the discussions that ***"it is the increased respiratory work that makes the patient short of breath".***
- The respiratory work is expressed as kg m/minute
- When the respiratory work raises from the normal value of about 0.3 kg m./minute to as high as 2-3 kg m. /minute, then dyspnea is usually present.
- The limit that leads to dyspnea varies with patient to patient. "Not every patient becomes dyspneic"
- In simpler terms when the effort required is increased dyspnea occurs.
- Chest diseases upset the respiratory mechanics and will increase the work load.
- Thoracic cage problems – kyphoscoliosis, ankylosing spondylitis, etc. reduce the chest wall compliance and increase the work load.
- Chronic bronchitis, asthma, emphysema lead onto *increased airway resistance, decreased dynamic compliance*. Air trapping occurs with increase in lung volume. Chest wall is gradually fixed in a position of inspiration.
- These patients compensate for the loss of elastic recoil of lung by actively contracting the abdominal muscles during expiration.

Clinical Significance of Resistance to Ventilation

- *The resistance to the flow of air in a tube is the pressure difference between the two ends at a given flow rate.*
- Under normal conditions of breathing the respiratory resistance comprises the force required to drive the air to and fro along the bronchial tree from the mouth to the alveoli.
- If a subject breathes through an endotracheal tube, then the extra breathing pathway must also be taken into account, because the respiratory muscles will have to do more work.
- A simple test is to ask a conscious person to breath through a small endotracheal tube (4 mm or 6 mm) held between his lips, when the extra effort required soon become apparent.
- With *increased resistance expiration is more difficult* because of increased intrapulmonary pressure with inadequate emptying of alveoli.
- This leads to increase in alveolar carbon dioxide and increased depth of respiration in an attempt to wash out carbon dioxide. *Expiratory muscles soon come into play.*
- Nearly every piece of breathing circuitry increases varying degree of respiratory resistance.
- The type of flow of gases makes a major difference, which depends upon the *diameter of tube, connecting pieces,* and the number.
- When the flow through a tube which is smooth and regular, it is ***"laminar",*** but when the rate of flow increases a critical velocity is reached at which the flow becomes ***"turbulent".***
- The practical significance is that ***the largest sizes tube that passes easily through the glottic aperture*** is to be

passed, as this is the narrowest portion of the respiratory tract between the lips and carina. Any size above 8 mm is acceptable for an average adult.

- It has been shown that, when adult is intubated with a 6 mm endotracheal tube, there is an enormous increase in the resistance to inspiration noted by the increased intrapleural pressure. Whereas when the same person is intubated with a 10 mm size endotracheal tube, the intrapleural pressure drops to almost normal (Figs 6.22 and 6.23).

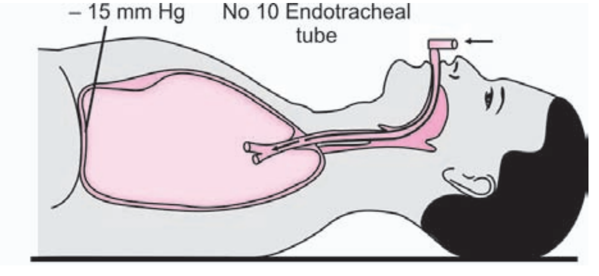


Fig. 6.22: A patient intubated with a 10 mm diameter endotracheal tube

❖ The 10 mm diameter endotracheal tube offers minimal resistance to breathing.

❖ However, even that artificial airway has caused added resistance as seen by an increase in intrapleural negative pressure.

- This clearly indicates that the size of the endotracheal tube significantly contributes for the resistance to ventilation. During mechanical ventilation, it is more significant in expiratory phase as expiration is a passive process.
- In children the cricoid ring is the limiting factor.
- Connectors should be of a ***wide bore*** and ***smoothly curved*** rather than sharply angled. There is laminar flow with least resistance in the curved connector and

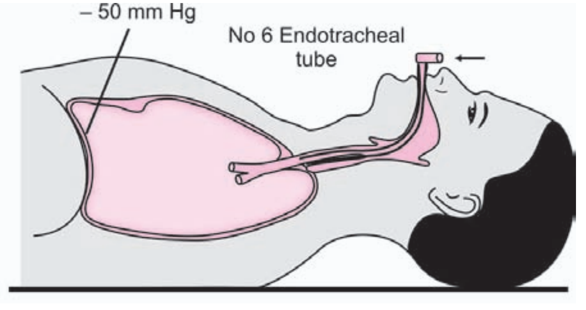
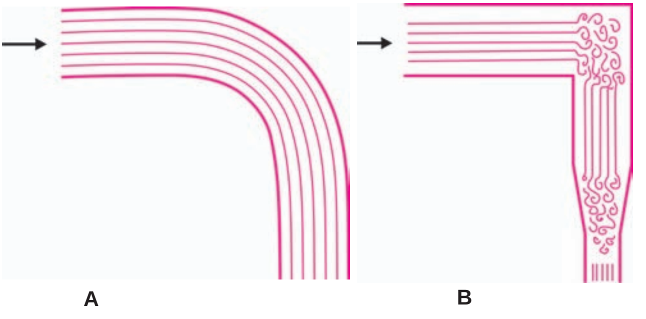


Fig. 6.23: The same patient intubated with a 6 mm diameter endotracheal tube

❖ The smaller size (6 mm diameter) endotracheal tube offers enormous resistance to breathing that the intrapleural pressure rises to – 50 mm Hg.



Figs 6.24A and B: (A) Laminar flow in curved tube, (B) Laminar flow become turbulent in right angle and at the narrowing

turbulent flow with increased resistance in the right angled connector (Fig. 6.24).

- Acute angles in any breathing circuitry are always avoided to prevent increase in resistance.

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CHAPTER 7

Mechanical Ventilation: Machine-assisted Breathing

- *Definition*
- *Intermittent positive pressure ventilation (IPPV)*
- *Types of artificial respiration*
- *The simplest ventilator*
- *The pressures related to respiration*
- *Normal pressure ranges during spontaneous respiration*
- *Physiological effects of positive pressure ventilation*
- *Manipulation of respiratory cycle in mechanical ventilation*
- *Possible modifications in each phase*

During normal respiration, when the respiratory muscles contract, the thoracic cage is expanded which causes an increase in the intrathoracic volume. This will create a subatmospheric pressure in the alveoli. Thus a pressure gradient is created between the upper airway and the alveoli which allow the air to move into the lungs. Expiration is normally a passive process and the natural elastic recoil of the lung tissue causes an increase in the intra-alveolar pressure to reverse the pressure gradient and allows air to flow out of the lungs. *Purpose of ventilation is to transport O_2 into blood, remove CO_2 from blood and maintain normal blood gas.*

DEFINITION

Mechanical ventilation is the use of a machine to induce alternating inflation and deflation of the lungs, to regulate the exchange rate of gases in the blood.

- Ventilation literally means *"movement of air"*
- In physiological terms it is the "movement of air in and out of lungs"
- The term *"ventilation"* is used to refer to *"respiration"* in physiology.

- Hence mechanical ventilation is *"Machine-assisted respiration or breathing"*

The primary difference between normal respiration and mechanical ventilation is the positive *pressure,* and it is applied to the airway *intermittently and rhythmically.* Hence it is named as Intermittent Positive Pressure Ventilation (IPPV).

Therefore, the basis is of mechanical ventilation is IPPV. All other sophistications are added subsequently on to it one by one to improve the quality of ventilation.

When artificial ventilation is thought of, the first thing that comes to our mind is the positive pressure ventilation instituted for an accident victim with cardiorespiratory arrest.

If suddenly some one collapsed and not breathing due to any reason, *the first step of resuscitation is to institute artificial respiration* to him and simultaneously check whether his heart is beating. Respiration is essential to oxygenate the blood and then the blood is pumped by cardiac compression. Pumping deoxygenated blood, serves no purpose.

- Artificial respiration means artificial ventilation.
- It is simply moving air in and out of lung artificially.
- *If the diffusion process in the alveoli and the perfusion to the lungs are normal, then the blood gas level may be maintained at normal range.*
- If at tissue level utilisation of oxygen is affected (internal respiration), artificial respiration may not be of help to maintain normal blood gas levels.

TYPES OF ARTIFICIAL RESPIRATION

Artificial respiration may be established in any one of the following ways.

- **Mouth to mouth breathing:** When nothing else is available, the rescuer can use *his respiratory apparatus* for breathing for the victim also. It is known as *"Breathe for two"*. The expired air which contains 16% oxygen

is forced into the patient's lungs by **positive pressure** generated by the rescuer in his respiratory apparatus (Fig.7.1A). This technique is aesthetically very unpleasant. Placing a handkerchief over the mouth of the victim may make it more acceptable.

- **Mouth to mask (Fig. 7.1B)**: The Laerdal pocket mask makes expired air ventilation more pleasant to perform. This is more hygienic and additional oxygen if available may be added through the nipple present on the body of the mask.
- **Mouth to airway:** These devices make expired air ventilation easy as the pharyngeal portion (body of the airway) helps to maintain a patent airway. Various types of airways may be used for this purpose. **Safar's airway** (Fig. 7.2) and **Brook airway** (Fig. 7.3) are very effective as there is blow tube in these devices which make expired air ventilation more effective and acceptable for the rescuer. The *Brook airway* has the additional advantage that it has *a unidirectional valve* which diverts the expired air from the patient away from the rescuer.
- A **Combitube** airway also can be passed into the mouth blindly, to reach the esophagus which has two tubes and two cuffs. One tube communicates through the perforations leading to glottic opening. After inflating the two cuffs available ventilation could be achieved through the tube which communicates with the glottic opening (Fig. 7.4).
- **Bag-Valve-Mask.** These devices have three components; a self inflating bag, a non- rebreathing (unidirectional) valve, and a face mask (Fig. 7.5). The mask can be applied to the face in the proper way and by squeezing the bag intermittently; the lung can be inflated with air allowing passive expirations. This may be considered as a very primitive method of instituting Intermittent Positive Pressure Respiration (IPPR).

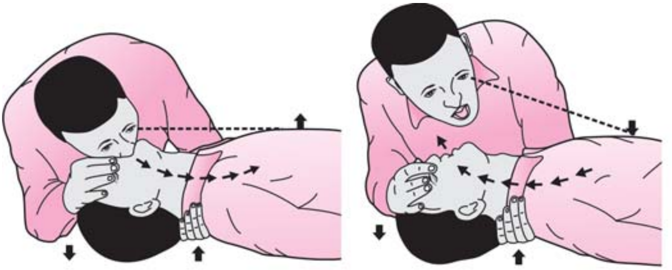

Fig. 7.1A: Mouth to mouth ventilation

Fig. 7.1B: Pocket mask for mouth to mask ventilation

The Simplest Ventilator

- The simplest ventilator will divide the minute volume into the number of breaths (tidal volume).
- A self inflating resuscitator bag such as **Ambu's bag** can be considered as a simplest and crude form of ventilator (Fig. 7.5).
- Any ventilator must have two components namely; the control mechanism (brain) and a driving force (muscles).

Fig. 7.2: Safar's airway

Fig. 7.3: Brook airway

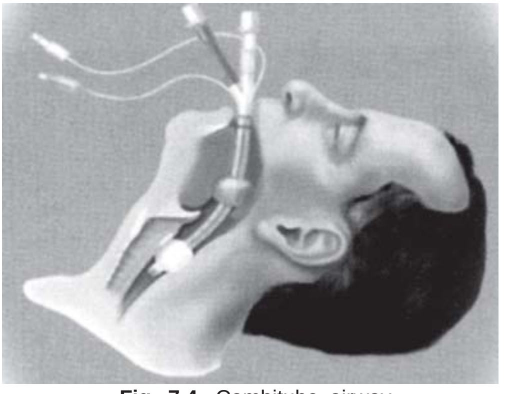

Fig. 7.4: Combitube airway

Fig. 7.5: Bag-Valve-Mask for IPPV

- Here the control mechanism and driving force belong to the operator.

East Radcliff Ventilator (East Radcliff Positive-Negative Respiration Pump)

This is discussed in detail here only because it worked on *very simple mechanical principles* and saved many lives. The ventilation is achieved by mechanical compression of a bellow intermittently by an electrically driven motor at a

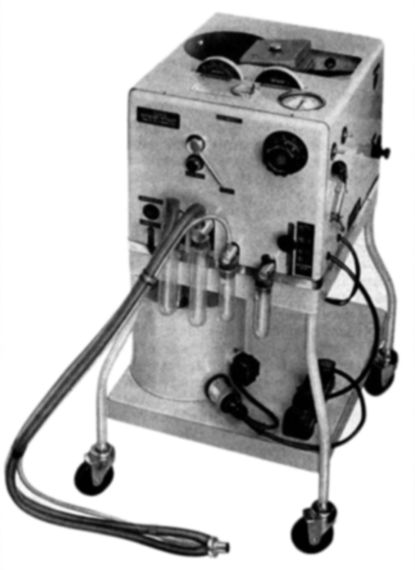

Fig. 7.6: East Radcliff positive-negative respiration pump

set volume, pressure, and rate. Truly a "Mechanical ventilator" indeed!

- This was probably the best tried and most widely used ventilator in the United Kingdom from 1956 (Fig. 7.6).
- It was particularly suitable for patients who are permanently dependent on artificial ventilation. A number of such patients have used simple models of the Radcliff ventilators in their own homes for up to 10 years (Fig. 7.7).
- One of the early ventilators which was in wide use in our country in early 1960s.
- It is known as *"positive–negative respiration pump"* because there is provision for applying negative

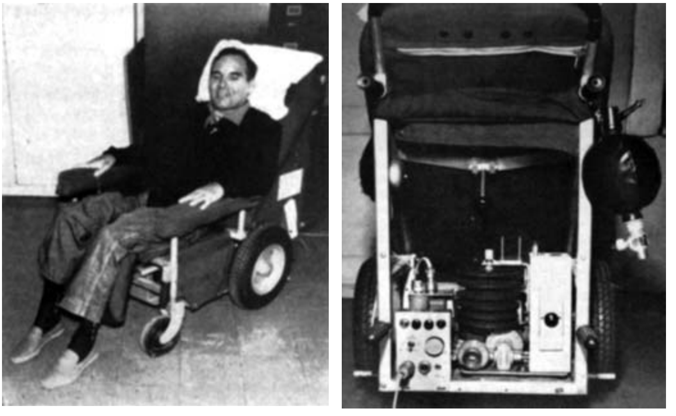

Fig. 7.7: A Radcliff ventilator used in a wheel chair at home

pressure during expiration to aid emptying lung. Later it was found to cause atelectasis.

- A robust machine which could continuously work for months without any problems.
- A concertina bellows works as the blowing mechanism.
- An electric motor drives to compress the bellows to force the air.
- The rate of ventilator is adjusted by a variable drive gear at rates of 12, 14, and 16 per minute.
- The inflation pressure could be adjusted by adding or taking out metal blocks of weight on the concertina bellows.
- A pressure gauge measures the inflation pressure.
- The tidal volume and minute volume is measured by the Wright's respirometer in the inspiratory limb (Not in the expiratory limb as in the newer ventilators).
- The motor is electrically operated, in the event of power failure a standby car battery provided in the ventilator takes over.

- In case of necessities, the ventilator bellows can be operated by cranking the handle provided in the machine.
- An electrically operated steam humidifier also is provided in the ventilator.

In this chapter, first we can just look into the changes that can occur in the respiratory mechanics when *positive pressure* is applied to airway to force air into the lungs.

Before going to the mechanics of positive pressure ventilation, we may briefly recall the mechanics and the pressure changes in *normal respiration* and then *compare it with the positive pressure respiration* for a better orientation.

REQUIREMENTS FOR NORMAL RESPIRATION

- Fresh air from atmosphere has to *flow into the lungs* during inspiration, to be exchanged at the alveolar–capillary membrane.
- During this process, the gases have to be *distributed uniformly* into all the *areas of lung* for exchange.
- The *perfusion* to the lungs must be *adequate* and *uniform* for good gas exchange.
- Now after the exchange, the air containing CO_2 that diffused out in the alveoli has to be exhaled during expiration.
- This process has to be repeated rhythmically.

The basic principle in pulmonary **gas flow** is that **a pressure gradient** is created so that gases travel from area **of higher pressure to lower pressure.**

THE PRESSURES RELATED TO RESPIRATION

In pulmonary physiology all pressures related to the ventilation are mentioned in **cm H_2O.**

Atmospheric Pressure

- This is the pressure exerted by the surrounding air on the earth's surface. This is **760 mm Hg** at sea level.
- In pulmonary ventilation, any pressure that equals the atmospheric pressure is mentioned as **0 cm H_2O.**
- Any pressure above atmospheric pressure is called *positive pressure* (supra atmospheric).
- Any pressure lower than atmospheric pressure is called *negative pressure* (subatmospheric).

Intra-alveolar Pressure (Palv)

- This is the pressure within the bronchial tree and the alveoli.
- At rest, (at the end of expiration) it equals to atmospheric pressure, i.e. **0 cm H_2O.**
- During inspiration, this pressure becomes negative, i.e. **– 1 to 2 cm H_2O.**
- During expiration, the intra-alveolar pressure becomes slightly positive, i.e. **+ 1 to + 2 cm H_2O.**

Intrapleural Pressure (Intrathoracic)

- This is the pressure within the pleural space.
- This is subatmospheric or negative because of the tendency of the lungs to collapse and causing a pull inwards.
- Elastic recoil of the lungs continuously exerts a pull on the thoracic wall.
- At rest (at the end of expiration) this pressure is negative, i.e. **– 5 cm H_2O**.
- During inspiration, this becomes more negative, i.e. **– 8 to –10 cm H_2O.**
- During expiration, it is less negative, i.e. **– 4 cm H_2O.**

Transpulmonary or Transmural Pressure

- This is the *pressure difference* across the lung; *intra alveolar – intrapleural pressure*; created entirely by the elasticity of the lung and it is always positive.
- The gradient increases during inspiration causing air flow into the lungs
- At rest it is **+ 5 cm H_2O.** (at the end of expiration)
- During inspiration, it is **+7 cm H_2O.**
- During expiration it is **+ 5 cm H_2O.**

THE PROCESS OF RESPIRATORY CYCLE

- *At the end of expiration*, we assume that the lungs are at resting condition. So at this point of time the *pressure within the alveoli is atmospheric* (**0 cm H_2O**).
- When a spontaneous inspiration is initiated, muscular effort is exerted by the contraction of the diaphragm and the external intercostals muscles. Inspiration is thus an active process and that requires the expenditure of energy.
- Contraction of these inspiratory muscles enlarges the thoracic cavity. The lungs expand because they are pulled out wards along with the movement of the thoracic wall. The lungs move with the chest wall because of the surface tension created by the small amount of fluid present between the visceral and parietal pleurae.
- The intra pleural negative pressure, which is normally present, becomes more negative on inspiration (Refer to Figs 6.3A and B in Chapter 6).
- Intra-alveolar pressure also becomes negative creating a gradient to atmospheric pressure. When alveolar pressure drops below atmospheric pressure, air flows

into the lungs because of its tendency to move to an area of lower pressure.

- In other words, the atmospheric pressure necessarily tries to equalize the pressure in the alveoli by allowing the air to flow into the tracheobronchial tree to fill the alveoli, as it is open to the atmosphere. Air simply moves from the higher pressure to the lower pressure. (Fig. 7.8, and Fig. 6.3 in Chapter 6)
- Inspiration continues till the intra-alveolar pressure equalizes with the atmospheric pressure.
- Expiration is a passive process which occurs because of the elastic recoil of the lung.
- When the contraction of inspiratory muscles ceases, the thoracic cage and the lungs recoil to their original size.
- Intrapleural pressure becomes less negative.
- Intra-alveolar pressure becomes slightly positive (+1 to +2 cm H_2O) which is enough to drive out the extra air in the lungs (Tidal air drawn in during inspiration) into the atmosphere. Thus expiration is effected till the intra-alveolar pressure equalizes with the atmospheric pressure (Fig. 7.8).

Normal pressure ranges during spontaneous respiration
(see Fig. 6.3 in Chapter 6)

Pressure	*Inspiration*	*Expiration*	*Resting*
Atmospheric (760 mm Hg)	0 cm H_2O	0 cm H_2O	0 cm H_2O
Intra-alveolar	**– 1 to – 2 cm** H_2O	**+ 1 to + 2 cm** H_2O	**0 cm** H_2O
Intrapleural	**– 8 to – 9 cm** H_2O	**– 4 cm** H_2O	**– 5 cm** H_2O
Transmural (Intra-alveolar – intrapleural)	**+ 7 cm** H_2O	**+ 5 cm** H_2O	**+ 5 cm** H_2O

At this point it is important to remember that, the increased negativity of intrapleural pressure is essential to bring air into the lung. Another important function of

this negative pressure is to promote venous return to the right side of the heart (preload) by expanding the great veins.

When an individual is placed on a positive pressure ventilator, the normally low intrathoracic pressures are disrupted and become positive. Positive pressure within the thorax affects the distribution of gases and may also cause hemodynamic embarrassment, primarily through a reduction in the venous return. This effect may be more pronounced in critically ill patients with poor cardiovascular reflexes.

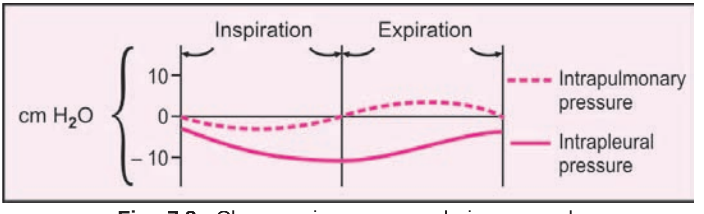

Fig. 7.8: Changes in pressure during normal inspiration and expiration

So far, the process of normal spontaneous respiration has been discussed, now it is apt to discuss the process of positive pressure respiration and how it is different in the changes of pressures produced.

The normal physiological respiration, energy is expended to contract the muscles of respiration. Contraction of muscles of respiration enlarges the thoracic cavity, which creates a negative pressure within the chest and results in the flow of air, at atmospheric pressure, into the lungs.

It would be ideal, if the same principle can work for the flow of air into the lungs in mechanical ventilation also. The negative pressure ventilators attempted to do so with lots of limitations and disadvantages. Hence negative

pressure ventilators have gone out of use. All modern ventilators work on the principles of positive pressure for driving air into the lungs. They apply supra-atmospheric pressure to proximal airway that forces air into lungs

- A mechanical device generates positive pressure and applies it on airway that forces air into the lungs. This is the inspiration (Fig. 7.9).
- Then the expiration is allowed to occur passively.
- Positive pressure is applied again to repeat the process of inspiration.
- Intermittently applied positive pressure on the airway to force air in out of the lungs and so it is known as "Intermittent positive pressure ventilation" **(IPPV).**

This is the basis of mechanical ventilation:

- The positive pressure ventilator uses a power source known as the ***drive mechanism***, to force air into the lungs during inspiration.
- As discussed earlier, the ***expiration*** is allowed to occur ***passively.***

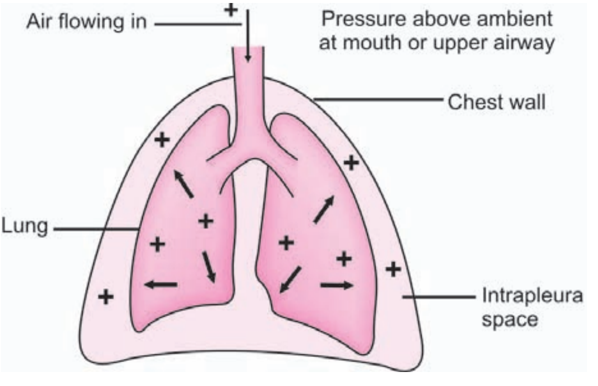

Fig. 7.9: Changes in the alveolar and intrapleural pressures during positive pressure inspiration

In spontaneous breathing, the respiratory cycle of "Inspiration, ending inspiration, expiration and starting a new inspiration" is not done with voluntary efforts. If a machine is to perform the respiratory cycle, it must be instructed, first what the component phases of the respiratory cycle are and second, how to carry out each of the phases as determined by the settings of the phase variables.

POSITIVE PRESSURE VENTILATION (IPPV)

Physiological Effects

The differences in the various pressures related to respiration in normal respiration and positive pressure respiration may be compared as follows.

Intrapulmonary Pressure (Alveolar)

- During both the phases of respiration (Inspiration and expiration) it becomes positive.
- Pressure difference between inspiration and expiration; normal spontaneous breathing it is about **2 cm H_2O** (During inspiration **– 2 cm H_2O** and during expiration **+ 2 cm H_2O)**.
- In positive pressure ventilation (IPPV), it is about **+ 10 to + 15 cm H_2O** during inspiration (inflation pressure) and returns to **0 cm H_2O** (atmospheric) at the end of expiration (Figs 7.10 to 7.12).

Intrapleural Pressure

- In normal spontaneous respiration it is always negative, more negative during inspiration **– 10 cm H_2O** and less negative at the end of expiration **– 5 cm H_2O** (Fig. 7.8).

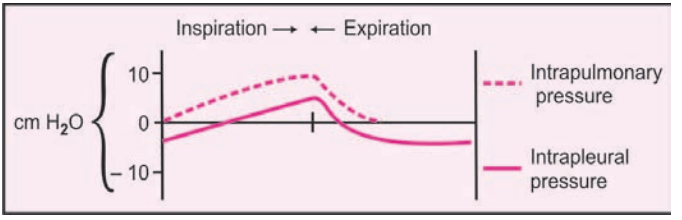

Fig. 7.10: Intrapulmonary and intrapleural pressures changes during positive pressure ventilation

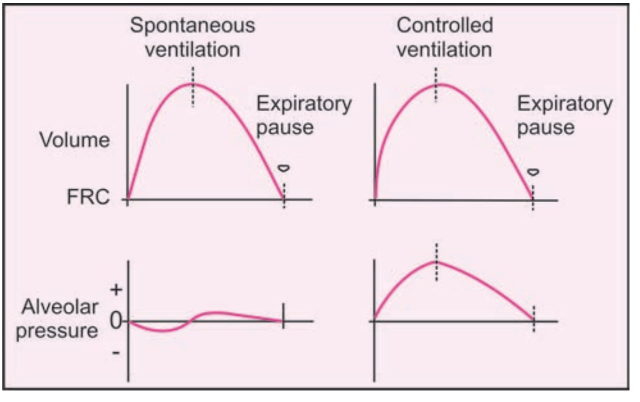

Fig. 7.11: Comparison of pressure volume curves in spontaneous and controlled ventilation

- In spontaneous ventilation alveolar pressure is very minimally negative during inspiration and becomes minimally positive during expiration.
- In positive pressure ventilation, alveolar pressure is positive during inspiration and expiration and drops back to baseline at the end of expiration.
- In both types of ventilation, it may be noted that the tidal volume delivered is the same.
- It can be correlated with Figure 7.10.

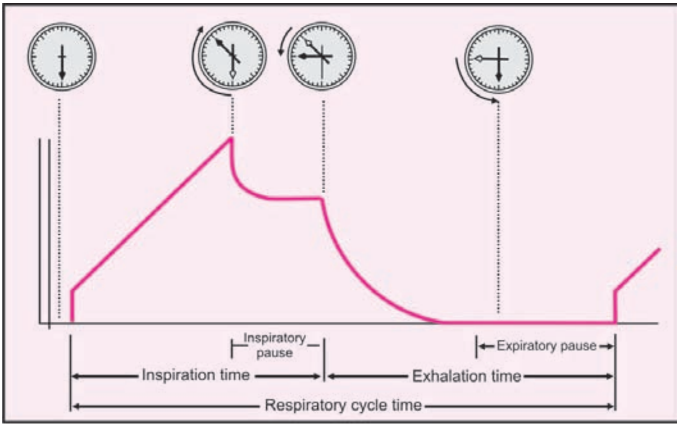

Fig. 7.12: Various pressures respiratory cycle in positive pressure ventilation in the form of graph (on the dial, the arrow with dark head shows the actual pressure and the arrow with hollow head shows the previous pressure)

- *End expiratory pressure:* It is 0 cm H_2O (atmospheric), just before the onset of inspiration.
- *Peak inspiratory pressure (PIP):* (15 cm H_2O). It is the maximum pressure reached at the end of inspiration.
- *Plateau pressure:* (10 cm H_2O). There is a slight fall of pressure during the distribution of gases in the lungs. From this it drops during expiration to baseline (0 cm H_2O).

- In positive pressure ventilation (I.P.P.V.), it is less negative at the start of inspiration and becomes positive during inspiration to reach the peak at the end of inspiration **– 5 to + 3 cm H_2O** and during expiration it rapidly falls, back to the original level of **– 5 cm H_2O** at the end of expiration (Fig. 7.10).

Lung Compliance

There is a significant reduction in the lung compliance occurs (about 50%) once IPPV is instituted. Though it is

a great disadvantage, for the merits of ventilation it is accepted.

Dead Space

There is a considerable increase in physiological dead space. This is secondary to the ventilation perfusion (V/Q) mismatch that occurs during IPPV.

Hyperventilation

A common problem in patients supported by mechanical ventilation is that they are over ventilated, which leads to respiratory alkalosis.

Respiratory alkalosis may cause:
- *Hypocapnoea* which depresses the respiratory center for any stimuli from periphery.
- Shifts the oxygen dissociation curve to left which is disadvantageous for delivery of oxygen to the tissues.
- Induces cerebral vasoconstriction which is advantageous for patients with head injury as it reduces the cerebral edema at the same time providing better oxygenation.
- There is a fall in intracranial tension and cerebral edema. These two factors are beneficial for patients with head injury.
- In patients with brain pathology, there may be "Inverse Steal" phenomenon where there will be vasoconstriction in normal area with vasodilatation in the affected area.
- Arterial hypotension is caused by reduction in sympathetic discharge.

Effects of IPPV on Cardiovascular System

- Because of converting the normal negative intrapleural pressure to positive side the negative pressure "Thoracic pump" which is likely to enhance the venous return (preload) to the right heart is abolished.
- It results in a fall in venous return to the heart and resultant fall in cardiac output.
- This fall in cardiac output and reduced sympathetic discharge cause hypotension
- Pulmonary blood flow is reduced because of the positive pressure (normal inflation pressure of 15 to 20 cm H_2O) exerted on the more compliant pulmonary vasculature. This results in a rise of pulmonary capillary pressure (normal is 13 cm H_2O) in turn leading to right ventricular strain.

When we consider the physiological changes in respiratory and cardiovascular systems due to IPPV, there are many aspects physiologically unfavourable, but there are certain undisputed benefits of mechanical ventilation to the patient.

Advantages of IPPV

- It improves gas exchange at alveolar capillary membrane.
- It takes over work of breathing (WOB) thereby allows rest for the muscles of respiration and prevents exhaustion.
- The respiratory depression due to any reason can be ignored as the ventilation is taken over by the ventilator.
- Heavy sedation and analgesia as required may be permitted without worrying about the possible respiratory center depression.

- IPPV may *open up collapsed alveoli and thereby cause recruiting more alveoli* to take part in gaseous exchange and improve oxygenation.
- Positive pressure ventilation may reverse pulmonary edema.

THE IDEAL VENTILATION CHARACTERISTICS

Ventilation

- Ventilation of the lung is the volume of air moving in and out of lung in the given time.
- *Volume = Flow × Time*
- *Tidal volume* is the flow of air into the lungs in the given time for inspiration
- *Minute ventilation = Tidal volume × Frequency (Respiratory rate)*
- *Ventilation = Flow × Time × Frequency*

The Factors that Modify the Ventilation in IPPV

Mechanical ventilation depends on characteristics of:

- ***Ventilator***
- ***Lung compliance***
- ***Airway resistance (LCAR)***

The lung compliance and airway resistance have been discussed in detail in the chapter on "Mechanics of breathing" (Chapter 6).

Problems Associated with Mechanical Ventilation

It has been established that a patient who is ventilated with mechanical ventilator is likely to encounter certain problems due to some of the variables. Commonly three types of injuries are possible.

1. Excess of O_2 in the inspired gases (High F_IO_2)

- O_2 radical mediated lung injury: Breathing oxygen when concentration over 60% for prolonged periods at atmospheric pressure may cause this damage probably due to inactivation of surfactant and damage to pulmonary epithelium. Free oxygen radicals are proposed to be the causative factor in alveolar damage.
- Breathing 100% oxygen may be harmful if continued for more than a few hours whereas 40% can be breathed indefinitely without problems. A characteristic picture is produced. (This has been discussed in oxygen toxicity in Chapter 5).

2. Excess flow of gases (*Barotrauma/volutrauma)*

- In fact this is stretch injury, *overdistending the alveoli* and is caused by excess flow of gases.
- If the alveolar injury or rupture occurs because of excessive pressure caused by the increased flow, it is known as *barotrauma.*
- If the excess flow has caused excessive peak inflating volume, it is known as *volutrauma.*
- Commonly both the components may be present.

3. Ventilator muscle overload:

- This problem arises when the patient's requirements and the ventilator settings do not match each other. Therefore, the patient-ventilator asynchrony is caused resulting in inadequate ventilation. This is sometimes called as patient *"fighting the ventilator"*
- This is a more complex phenomenon which can be managed by understanding the patient's needs and accordingly making the ventilator settings.

- Giving more sedation to make the patient accept the ventilator or paralyzing the patient with NMB drugs may also help overcome this problem.
- This asynchrony if occurs, may cause delay in weaning process.

Muscular Atrophy of Diaphragm

- If a patient is on mechanical ventilation for more than 40 hours, there is a reduction in the muscle mass of diaphragm. It suggests that lack of activity in diaphragm leads to muscular atrophy.
- Therefore, whenever possible, minimal amount of work is to be maintained for diaphragm to prevent reduction in the diaphragmatic strength and endurance.
- Using *Assist/control mode* may be useful in preventing respiratory muscle atrophy.
- Outcome of ventilation therapy depends upon the reversibility of the underlying disease that necessitated the therapy.

Setting a Respiratory Cycle in IPPV

The respiratory pattern required for a particular patient could be achieved by setting the variables suitably. The normal settings are given below.

- Inspiration : Expiration ratio: **1 : 2**
- Inspiration: **1. 75 Seconds**
- Expiration: **2. 50 Seconds**
- Expiratory pause: **0. 75 Seconds**
- Each cycle: **5 .00 Seconds**
- Respiratory rate: **12 per min (60/5 = 12)**

Suitable modifications have to be made according to the need of the individual patient.

Manipulation of Respiratory Cycle in Mechanical Ventilation

As discussed earlier, if a machine is to perform the work of respiration, it has to be instructed how to do it and when to do it. Therefore, for the sake of construction of a ventilator, the respiratory cycle is *divided into four phases so that the machine is instructed to perform the cycle in the very same pattern as normal respiration.*

It is further possible to make suitable modifications in each of the phases as required for improving the quality of respiratory function.

A respiratory cycle created by a machine is known as a **"Mechanical breath"**. It is necessary to know how these four phases are managed in a mechanical breath. The following discussion will deal with the phases and their management in a mechanical breath.

The Four Phases

- Inspiratory phase
- Change over from inspiration to expiration (cycling)
- Expiratory phase
- Change over from expiration to inspiration (Initiation of inspiration)

All ventilators are designed with these four phases as basic features. *It has to be remembered that what ever is done in the* ***form of modifications*** for the improvement of ventilatory function, could be done ***only in any one of these phases*** and *not out of it* (Fig. 7.13).

This figure depicts the four phase of a mechanical breath and the possible modifications in each phase.

Phase 1. Inspiratory phase:

- The primary function of the ventilator in this phase is to drive gas into the lungs by applying positive pressure to the proximal airway.

- In positive pressure ventilation, the inspiration usually starts from the baseline after a normal expiration. The baseline is atmospheric pressure (0 cm H_2O), that is when the lung has the FRC.
- As the pressure rises, the flow into the lungs continues till it reaches the set level as shown in the pressure–time wave form in Figure 7.13. This is the inspiratory phase.
- Once the desired limit is reached, the ventilator stops the inspiration and allows the passive expiration to follow. This is commonly known as **"Cycling."**
- The inspiratory phase can be ended by any one of the variable such as a preset level of ***Pressure, Volume, Time*** or ***Flow*** as we desire.
- The other modifications that can be made in this phase are ***"Pressure support"*** (PSV), ***"Pressure Controlled"*** (PCV), ***"Proportional Assist Ventilation"*** (PAV) and others will be discussed in detail in the next chapter.

Phase II. Change over from inspiration to expiration: **Cycling** (Fig. 7.13)

- The cycling is simply ending the inspiration and allowing the passive expiration.
- As it is discussed earlier, the cycling may be done with the four variables as, a preset level of ***Pressure, Volume, Time,*** or ***Flow*** as we desire.

Phase III. Expiration:

- Expiration is a passive process in IPPV also.
- Once the positive pressure on the airway is stopped, the lung empties the gases into atmosphere.
- The modifications that can be made in the expiratory phase such as PEEP, BIPAP, etc. will be discussed in detail in the next chapter (Fig. 7.13).

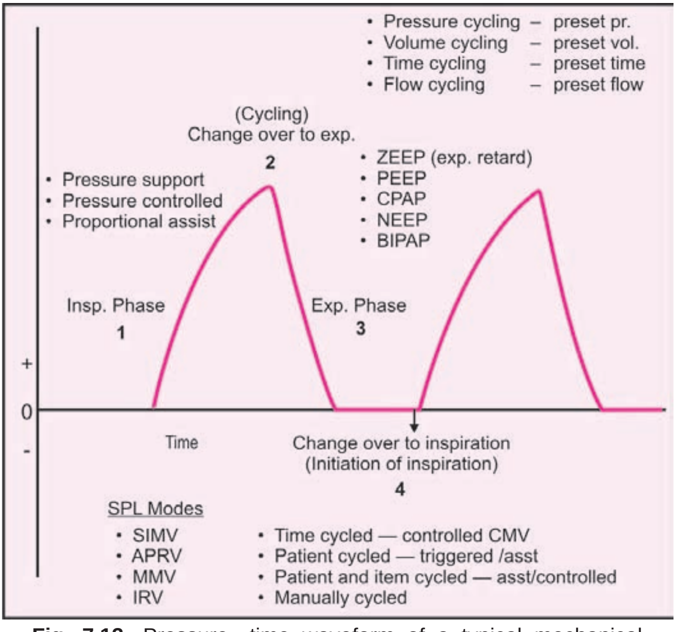

Fig. 7.13: Pressure—time waveform of a typical mechanical breath and the phases

- Inspiratory phase **1.**
- Changeover from inspiration to expiration (Cycling) **2.**
- Expiratory phase **3.**
- Changeover from expiration to inspiration (Initiation of inspiration) **4.**

Phase VI. Initiation of inspiration: Changeover from expiration to inspiration:

- Once the expiration is completed and expiratory pause is also allowed, the next inspiration will be initiated by setting some variables.
- Usually it may be **preset time** (CMV) or **patient's inspiratory attempt** (Assist) or **combination** of patient attempt failing which by the preset time (Assist/ Control) or it can be initiated **manually** also. This phase

also sometimes mentioned as cycling as time cycled, patient cycled, manually cycled, etc. (Fig. 7.13).

Possible Modifications in Each Phase

The possible simple modifications that could be made in each phase of respiratory cycle may be discussed here. Instead of starting from the inspiratory phase, with a small deviation, *"changeover from inspiration to expiration"* **(Cycling)** may be discussed first.

This is because, the mechanism involved in this phase is of considerable practical significance. In this way it is convenient to discuss and easier to understand.

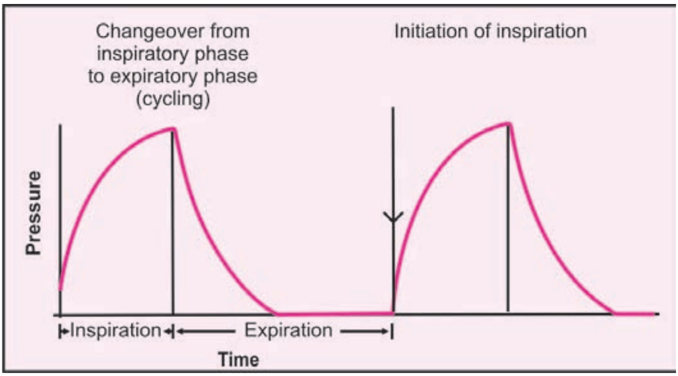

Fig. 7.14: Pressure—time curve in positive pressure ventilation showing four phases

Changeover from Inspiration to Expiration

This process is referred to as "**Cycling".** The mechanism by which the cycling is effected is of paramount importance (Figs 7.13 and 7.14).

- Once the ventilator starts the inspiration and delivering the gas into the lungs, at the set target point, when the desired volume of gas delivery is completed, or when the desired pressure is reached, the inspiration is completed. Now the flow of gas is to be stopped and expiration is to be allowed.
- The ventilator could be instructed to terminate the inspiration and for that four variables could be used as limits.

Types of Cycling—Mapleson 1969

Cycling: Changeover from Inspiration to Expiration

- **Pressure** cycling – limiting inspiration at a preset pressure
- **Volume** cycling – limiting inspiration at a preset volume
- **Time** cycling – limiting inspiration at a preset time; this can have two other variables;
  - **Pressure** limited
  - **Volume** limited
- **Flow** cycling – limiting inspiration at a preset flow

Most of the modern ventilators incorporate all these in one set up with computer control and suitable options could be combined in one setup.

Pressure cycling

- The machine is instructed to stop inspiration as the set pressure is reached.
- The inspiration ends when a *preset pressure is reached.*
- The *volume delivered*, *the flow rate*, and *the inspiratory time* are variable.

- Both the duration of inspiration and the volume of gas delivered will decrease when the compliance is reduced.
- Pressure cycled ventilators have one advantage; leak compensation, provided it is a minor leak. The inspiration goes on till the set pressure is reached despite the leak. This will allow the volume, flow, and time that are sufficient to ventilate the patient.

Volume cycling

- The machine is instructed to stop the inspiration when the set volume is delivered.
- The inspiration ends when the ventilator *delivers a preset volume.*
- *The time* required for delivering the volume, *the flow rate,* the *pressure developed* are all variable.
- When the compliance is low, high airway pressure may result.
- A volume cycled ventilator may not always deliver the set volume to the patient.
- *Leaks in the system*: the ventilator does deliver a preset volume, but only a part of it reaches the patient as the rest leaks out.
- ***Compression volume:*** part of the volume leaving the ventilator is lost due to the expansion of flexible tubing of the breathing circuitry.
- This volume depends upon the *pressure generated* and *the internal compliance* of the system.
- If the tubing is fairly rigid, the amount of gases compressed is approximately 1 ml/cm H_2O per liter of available space.
- Thus if the ventilator has a rigid delivery system which has an internal volume of 4 liters, 4 ml of gas would be compressed for each cm of pressure.

- The *compressibility factor* in this case is 4 ml/cm H_2O. If the system is more distensible, the factor is greater, about 4.5 to 5 ml/cm H_2O.
- Thus compression volume can be a significant factor, if the compressibility factor is large and the tidal volume (VT) is small.
- If the *compressibility factor* is 5 ml/cm H_2O and tidal volume is 300 ml, half of the volume (150 ml) can be compressed into the system if a pressure of 30 cm H_2O is applied

Time cycling

- The machine instructed to end inspiration at the set time.
- *A time-cycled* ventilator ends inspiration once the *preset time* has elapsed irrespective of the pressure reached or volume delivered.
- However most of the time-cycled ventilators are made in such a way that they are also either ***volume*** or ***pressure*** limited.
  - *Time-cycled, volume limited* ventilators deliver a set volume within a set time.
- The flow rate varies and also the pressure.
  - *Time-cycled, pressure limited* ventilators achieve a preset pressure which is maintained for a preset time.
- The *airway pressure attained*, *the inspiratory flow*, and *the tidal volume* are all variable.
- The volume delivered varies depending on the pressure and the patient's compliance.
- Such a system is of advantage in the ventilation of infants in whom a deliberate leak is maintained around the tracheal tube in an effort to avoid laryngeal trauma due to a tight fitting tube.

- For a given pressure, doubling the compliance will result in double the volume delivered.
- If the applied pressure does not equilibrate in the alveoli, because of excessive airway resistance or inadequate inspiratory time or both, the tidal volume (V_T) may vary greatly.
- If the patient actively breathes, the tidal volume (V_T) may increase.

Flow cycling

- The machine can be instructed to stop inspiration when the set flow of gas is reached.
- In this, inspiration ceases when the inspiratory *flow has fallen to a preset level.*
- This is not a common type of cycling. This is available in Manley Servovent ventilator (see Pressure support ventilation on Page 302).

Of these types of cycling, **Volume cycling** and **Time cycling** are most commonly used.

Expiratory Phase

Once the inspiration is completed by the machine by the set cycling, the inflation of lung stops. At this point the expiration will start. (Change over from inspiration to expiration).

Expiration is normally a passive process. The ventilator only has to allow the lungs to empty the tidal volume. However, some innovations are employed in this phase in an effort to improve gas exchange.

At the end of expiration, the intra-alveolar pressure drops to atmospheric. It is otherwise called as zero pressure. (0 cm H_2O) (Fig. 7.12).

The end of expiratory phase could be modified if needed. Depending upon the end of expiratory phase, a ventilator can be classified to have one of the following,

- Zero end expiratory pressure (ZEEP)
- Negative end expiratory pressure (NEEP)
- Positive end expiratory pressure (PEEP).

Constant Atmospheric Pressure (ZEEP)

- Here, the lungs are allowed to empty passively into the atmosphere, so the alveolar pressure drops to 0 cm H_2O (atmospheric).
- *All ventilators must have this option.*
- The duration of actual expiration, i.e., the time taken by the lungs to completely empty the tidal volume is proportional to the product of pulmonary compliance and total resistance.

In this type of expiration where the end expiratory pressure is atmospheric (ZEEP), one modification was introduced to improve the ventilation. That is *"Expiratory Retard"*.

Expiratory Retard

A modification by applying a "retard cap" offers resistance to expiration and prolongs it (Fig. 7.15).

The mechanism and advantages of retard:

- A variable resistance fits tightly on the expiratory port. Expiration against this artificial resistance mimics the *"pursed lip breathing"* of patients with chronic obstructive pulmonary disease (COPD).
- This facilitates emptying of the lungs by *'splinting'* the airways open. This reduces premature airway collapse during expiration and prevents air trapping.

Disadvantages:

- This resistance to expiration, which may facilitate emptying of the lungs up to a certain limit, can have the just opposite effect beyond a certain point.

- If the next inspiration starts before the exhalation is complete, then air trapping occurs. *Progressive alveolar distension and barotrauma* may result.
- Increase in mean intrathoracic pressure which reduces the venous return and cardiac output.

Because of theses reasons, the expiratory retard is no longer in use now, but it is discussed here only to make it clear how innovations were added on to the basic ventilation and then eliminated because of demerits.

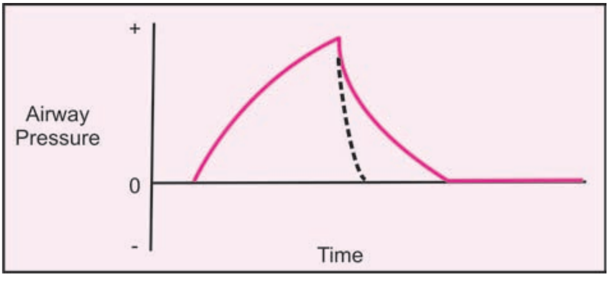

Fig. 7.15: Airway pressure waveform of an expiratory retard

❖ The interrupted lines indicate the courses of the waveform in the absence of retard.

Negative End Expiratory Pressure (NEEP)

- Here, a negative pressure is applied to the airway during the expiratory phase throughout the expiratory pauses till the next inspiration is started (Fig. 7.16).
- Usually the negative pressure is applied by attachment of a Venturi to the exhalation port.
- Though it reduces the mean intrathoracic pressure, and improved venous return and cardiac output, it *caused progressive collapse of the alveoli*.
- For this reason the NEEP is no longer used.

Here again it is discussed to emphasise how older innovations added to primary positive pressure ventilation have been eliminated.

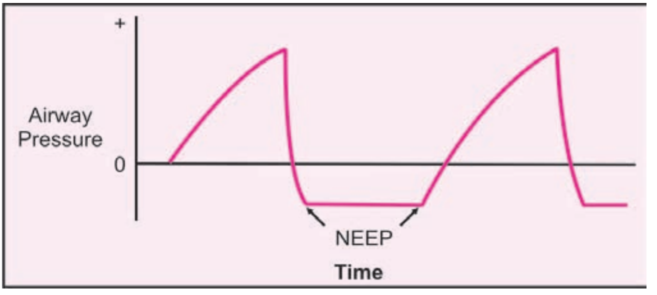

Fig. 7.16: The pressure wave form of NEEP

Constant Positive Pressure (PEEP)

- In this, the airway pressure during expiration is not allowed to decrease to atmospheric pressure, i.e. 0 cm H_2O. Instead, it will be maintained at a positive level.
- Thus a positive end expiratory plateau (PEEP) is produced (Fig. 7.17A).
- PEEP is produced by a *"threshold resister"* applied to the expiratory pathway.
- This differs from the expiratory retard in two ways;
  - With retard, in given time, the airway pressure falls to atmospheric, while PEEP implies *a positive end expiratory plateau.*
  - Pressure falls gradually when retard is used. However, in PEEP (at least in an ideal PEEP), there is *no resistance to expiration till the preset PEEP level* (the "threshold") is reached (Fig. 7.17B).
- Ideal PEEP, in which there is no resistance at all to expiration till the set level of PEEP is reached, is not achieved with any of the currently available devices.

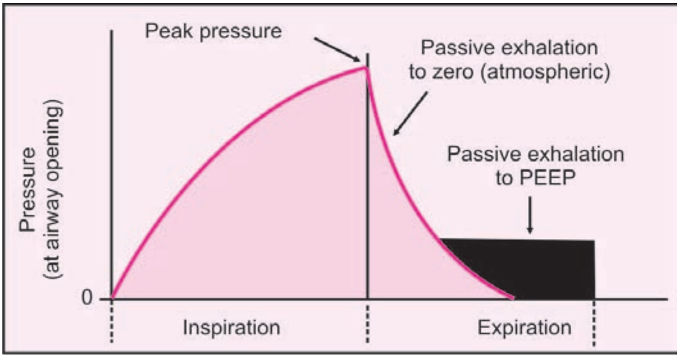

Fig. 7.17A: The mechanism of PEEP

- ❖ Note that in normal respiration at the end of expiration the pressure touches zero.
- ❖ In PEEP, the pressure at end of expiration does not touch zero, but stays higher.
- ❖ The area marked in black is the PEEP.

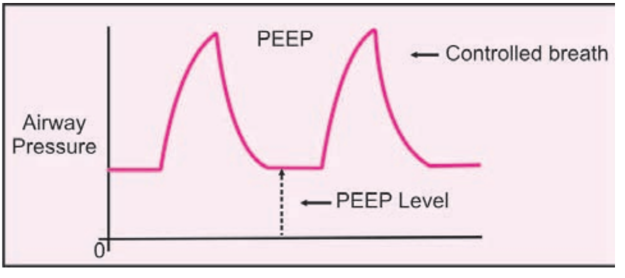

Fig. 7.17B: The pressure wave form of PEEP

- PEEP devices may be *internal* or *external*. A PEEP device is called internal when the device is incorporated within the ventilator. The external devices are attached to the expiratory port.
- When PEEP is used with spontaneous ventilation, it is called Continuous positive airway pressure (CPAP) (Fig. 7.18).

- In CPAP, the pressure in the patient's breathing circuitry is elevated to a level slightly above the ambient.

Disadvantages:
- Increased interstitial and alveolar water content
- Increased ADH

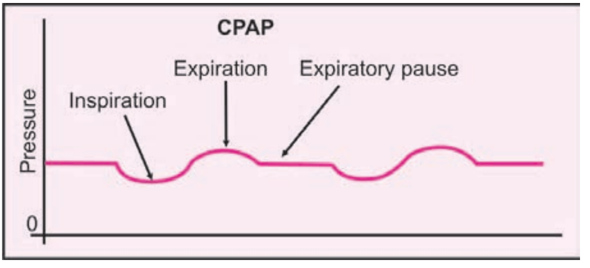

Fig. 7.18: Airway pressure time waveform CPAP—Continuous positive airway pressure

Initiation of Inspiration

So far "Cycling" and the types of cycling, expiratory phase, and the modifications that can be done on that have been discussed.

Once the expiration is completed, there is an expiratory pause before the next inspiration. In mechanical ventilation, the initiation of inspiration can be done by the machine in many ways as instructed, according to the requirement of the patient.

Depending on the way in which the inspiration is initiated, they are classified into *four types*. Sometimes the terms *"triggered on"* or *"cycled on"* are used in place of initiation. These two terms "triggering and cycling" may cause some confusion and the meaning has to be conceived correctly.

- **Time-cycled** (Control mode) (Controlled Mechanical Ventilation) (CMV)

- **Patient-cycled** or Triggered (Assist mode)
- **Patient/Time-cycled** (Assist/Control mode) (A/C)
- **Manually** cycled.

Time-cycled (Control Mode) (CMV)

- Positive pressure breaths are delivered automatically by some *timing mechanism* regardless of patient's effort. (Fig. 7.19)
- The mechanical breathes are at *specific time interval as set in the machine*.
- This mode is suitable for patients who are totally apnoeic and have no efforts of respiration.

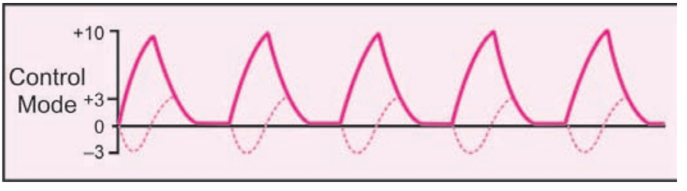

Fig. 7.19: Airway pressure – time waveform in 'control mode' ventilation

❖ The faint dotted line indicates the normal spontaneous breathing, if the patient breathes.

Patient-cycled or Triggered (Assist Mode)

- Here, a small negative pressure generated in the airway by the patient's spontaneous inspiratory effort '*triggers*' a mechanical breath (Fig. 7.20).
- The number of breaths delivered by the ventilator thus depends on the patient's spontaneous respiratory rate.
- Usually the degree of patient effort (by the fall of airway pressure below the end expiratory pressure) required to trigger the mechanical breath is known as *"sensitivity"* and that is *adjustable.*

- Pressure support ventilation (PSV) is a modification of this system; mechanical augmentation of spontaneous breaths up to a preset level of positive airway pressure as long as patient demand exists. The patient thus retains the control of the rate.
- *If the patient stops making any attempts of breathing, the triggering will be lost and the machine will not deliver a breath.*
- Because of the danger that the machine will stop delivering the mechanical breaths if the patient becomes apnoeic, the assist mode is no longer used alone, but is combined with control mode as Assist/Control mode.

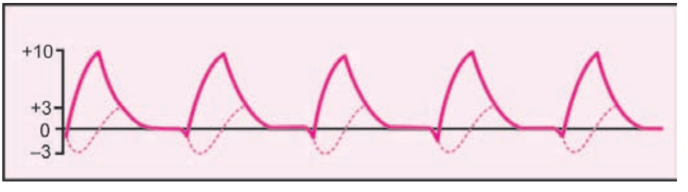

Fig. 7.20: Airway pressure – time waveform of Assist mode

❖ The small negative deflections at the start of the mechanical inspirations are the patient's inspiratory efforts which trigger the mechanical breath.

Assist-Control Mode (A/C)

- In fact it is a combination of *'control mode'* and *'assist mode'* for obvious benefits it can offer to the patient. (Figs 7.21 and 7.22)
- **"Assist"** refers to the patient triggered breaths being supplemented by the machine.
- A sensing device detects the patient's efforts by the fall in airway pressure below the end expiratory pressure and initiates the patient's breath.
- This fall in airway pressure required to trigger a breath is referred to as sensitivity and is adjustable.

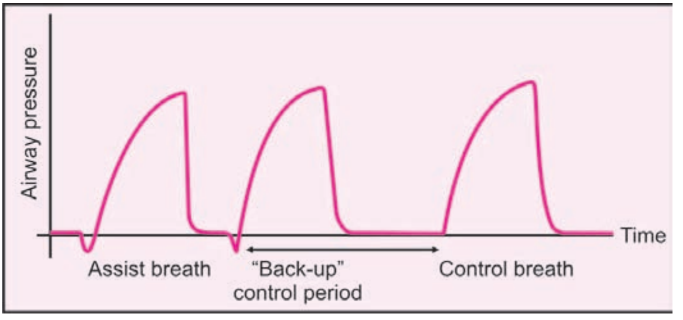

Fig. 7.21: Airway pressure time wave form in assist-control mode

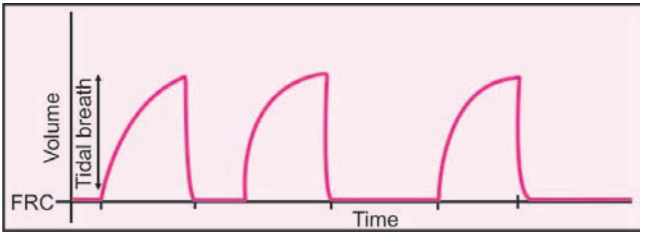

Fig. 7.22: Volume graph in assist-control breath

- Note that the first two breaths are patient triggered breaths and the third one is the mechanical breath delivered by the machine in the absence of trigger.
- The volume delivered is the same in patient triggered and mechanical breaths.

- **"Control"** refers to the preset (back up rate) operational during assist-control ventilation.
- If the patient on this mode fails to trigger a spontaneous breath with in the predetermined cycle period, then the machine will deliver a mechanical breath.
- If the spontaneous rate exceeds the control back up rate, no control breaths will be delivered, the machine will work as in assist mode.

Manual Cycling

- Manual cycling is usually available as an additional facility when the operator wants to deliver a breath independent of the patient's or ventilator's rate.

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CHAPTER 8

Mechanical Ventilator: Basic Concepts

- *Definition of a ventilator*
- *Classification of ventilators*
- *Basic concepts of a mechanical ventilator*
- *Features of a basic ventilator*
- *An ideal ventilator therapy*
- *Ideal initial setting*
- *Positive end expiratory pressure (PEEP)*

It is enough if the physician knows that the inspiratory effort of the patient creates a negative pressure in the airway which is sensed by the sensing device of the ventilator to open the demand valve to allow the inspiratory flow. There is no point in trying to know how this sensor works; as this is does not fall into our purview.

DEFINITION

There are very many definitions put forward by various authors. That way, a ventilator may be defined as,

- *"A device which can artificially maintain the respiration for a patient who cannot do it himself"*
- *"A device which causes bulk movement of gases in and out of lungs and takes over or assists the function of respiratory muscles".*

Based on the concept of construction a ventilator may be defined as,

- *"A ventilator is a machine – a system of related elements designed to alter, transmit and direct the applied energy (pneumatic power or electricity) in a predetermined manner to perform a useful work,* ***to augment*** *or* ***replace*** *the patient's* ***muscles*** *in performing the work of breathing".*

These are all many definitions of a mechanical ventilator. Whatever way we try to define, it simply means

that the patient's respiration is either fully taken over or assisted by a machine.

Basically, any ventilator must have two components for accomplishing artificial ventilation.

A control mechanism (a **brain):** To command what to do for ventilating the patient.

A driving mechanism (a **muscle):** To carry out the work of ventilation as it is ordered by the control mechanism (brain).

CLASSIFICATION OF VENTILATORS

Air movement into the lungs is provided by the contraction of respiratory muscles producing a subatmospheric (negative) intrapleural and intra-alveolar pressures. Thus formed pressure gradient between the upper airway and the alveoli allows the air to move into the lungs. Expiration is normally passive and the natural elastic recoil of the lung tissue causes an increase in the intra-alveolar pressure to reverse the pressure gradient and allows air to flow out of the lungs.

Mechanical ventilation is provided either by applying *a negative pressure around the chest* or by generating *a positive pressure* above the atmospheric pressure *to the upper airway.*

Although negative pressure ventilation does not require endotracheal intubation, it cannot overcome substantial increases in airway resistance or decrease in pulmonary compliance, and it also limits the access to the patient.

So by concept of construction and by priority in development, the mechanical ventilators are broadly classified into two groups, namely ***Negative pressure ventilators*** and ***Positive pressure ventilators.***

TYPES

Negative Pressure Ventilators

Negative pressure is applied on the thorax or abdomen. Eg: Cabinet ventilator (Iron Lung or Tank ventilator), Cuirass ventilator.

Positive Pressure Ventilator

- Positive pressure is applied to the proximal airway.
- Surface of the chest wall is in ambient pressure.

Most forms of mechanical ventilation intermittently apply positive airway pressure at the upper airway. During inspiration, gas flows into the alveoli until alveolar pressure reaches that in upper airway. During the expiratory phase of the ventilator, the positive airway pressure is removed or decreased; the gradient reverses, allowing the gas flow out of the alveoli.

The negative pressure ventilators now have only historical interest and their development has been discussed in the chapter on history (Chapter 2).

In modern times all the ventilators available are positive pressure ventilators, applying the basic principle of ***Intermittent Positive Pressure Ventilation (IPPV).***

Hence hereafter in all the discussions, the term 'ventilator' will mean only a positive pressure ventilator.

BASIC CONCEPTS OF VENTILATORS

- The advent of microprocessor technology allows a single ventilator to produce *any number of output waveforms, some as limitless as the operator's imagination*.
- *However, a simple ventilator may be a minute volume divider*. Simply minute volume can be divided into number of

tidal volume and delivered to the lungs rhythmically as the number of breaths per minute.
- In acute respiratory failure, the self-inflating resuscitator bag is used for ventilating the lungs by manual compression. As discussed earlier, this equipment may be considered as a primitive ventilator.
- In Intensive Respiratory Care Units (IRCU), highly sophisticated versatile machines controlled by microprocessors, with several modes of operation which incorporate monitors and alarms are in use.
- This shows a big gap between *simplicity* and *sophistication.*
- If the lungs are ventilated efficiently, the purpose is served and the life can be saved.
- If there is no ventilation, life cannot be sustained – so, any kind of ventilation without causing homodynamic instability is acceptable. This aspect has been elaborated in the previous chapter.
- In anesthetic practice it takes over the ventilation during anesthesia.

THE FEATURES OF A BASIC VENTILATOR

The ideal ventilator must have some basic features that are considered essential for a useful clinical application. They may be considered below.

The different variables with the desired range of each one is given below. There must be provision for the following,
- Inspired **O_2:** Up to **100%.**
- Inflation pressure: From **10 cm** to **40 cm** H_2O
- Cycles (respiratory rate): From **10** to **50**/min
- Tidal Volume: From **200 ml to 1500 ml,** ideally starting from **30 ml** onwards for use in pediatric patients.

- Monitoring tidal volume: Measured and monitored constantly.
- Ideally expired tidal volume is measured which is more accurate.

The other desirable features of a good ventilator may be:

- It must be simple in design and the controls must be such that they are *easy to operate*.
- It must be able to provide adequate ventilation *for all types of patients.*
- It must have *bacterial filters* at ventilator patient interface to prevent transfer of infection either side.
- It must have provision for easy *cleaning* and in case of necessity; the breathing circuitry and its components must stand *sterilization* by common methods like autoclaving and ethylene oxide sterilisation.
- It must have all the necessary alarm systems.

In the construction of the mechanical ventilators, in general **three** basic aspects are considered.

1. **Power input**
2. **Control scheme** (including power transmission or conversion)
3. **Output** (pressure, volume, and flow waveform).

Power Input (Driving Force)

The driving force of a ventilator is the force that generates the pressure sufficient to force the air into the airway-usually through the endotracheal tube to ventilate the lungs.

How? *"One form of energy (pneumatic power or electricity) is transmitted or transformed in a predetermined manner."*

Why? *"To augment or replace patient's muscles in performing the work of breathing."*

Usually any one of the two commonly used energy sources is employed here also for driving the ventilator. Sometimes one source is converted into the other in the ventilator and is used for driving it.

- Pneumatic power (compressed Air or O_2 from a cylinder or pipeline)
- Electric power
- Combined power.

Pneumatic Power

- Compressed air or oxygen from a cylinder or from the pipeline may be used for driving the ventilator usually at 50 to 60 lbs/square inch (psi).
- In the ventilator the pressure is regulated by reducing valves, needle valves, or venturi mechanisms.
- Technically, any ventilator that works on gas pressure would be Fluidic. Fluidic includes (1) *Pneumatics* (with moving parts) and (2) *Fluerics* (without moving parts).

Electric Power

- Here electric power is required for operating the ventilator.
- Oxygen supply is required only for increasing inspired oxygen concentration (F_IO_2). Even if the oxygen supply fails, the ventilator continues to deliver atmospheric air.
- Electricity is used to drive a mechanism (piston pump or alike) to be used for driving the ventilator. This type of ventilator is not common today.

Combined Power

- Here electricity is used to operate an air compressor that compresses air in to a cylinder from which the

compressed air is used as a pneumatic power for driving the ventilator. Most of the modern ventilators have a built in compressor.

- Some ventilators use both pneumatic and electric power sources. Usually compressed air or oxygen provides the driving force. The electrical power does the phasing and timing and controls the alarm systems.

Availability of uninterrupted compressed gas supply or electrical power is a point of importance, but now most of the modern ventilators come with many hours of battery backup for power.

Control Schemes

Control Variables

The following variables may be modified and controlled according to the requirement of the patient in need of support.

- Pressure
- Volume
- Flow
- Time.

Phase Variables

The following phases can be modified and controlled as required. For example *the base line may* be allowed to become *atmospheric* or it can be kept *above atmospheric pressure as in PEEP.*

- Trigger
- Limit
- Cycle
- Baseline.

Conditional Variables

According to the clinical situation the mode of ventilation can be chosen and instituted.

- Modes of ventilation (which will be discussed in the next chapter).

Output

Displays

The displays of the following characters are possible in panel to monitor the performance of the ventilator, as we desire.

- Waveform
- Pressure
- Volume
- Flow.

Alarm Systems

Alarms are essential components of any life supporting and monitoring device and hence for any ventilator also. Therefore, various alarms are incorporated in a ventilator to warn about any malfunctioning of the ventilator with regard to all the three basic aspects namely, Power Input, Control Schemes, and Output.

- Input power alarms
- Control circuit alarms
- Output alarms.

The above said description briefs the overall functioning modality of any modern ventilator. It is simple enough for us to understand how a ventilator functions, and not for us to memorize.

The simple basic principle and components of a modern microprocessor controlled ventilator can be briefed in the Figure 8.1.

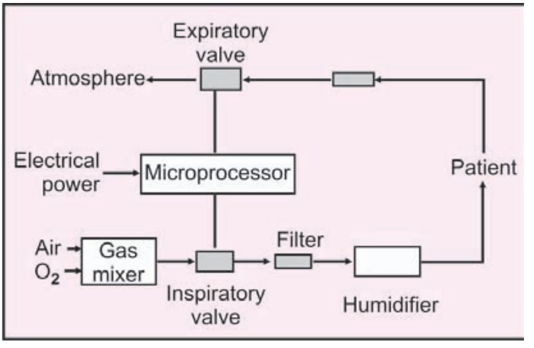


Fig. 8.1: The basic anatomy of a modern ventilator

"The user must know what the machine can do and not how it does it"

Hence simplifying all the above aspects in construction of a ventilator only two components are considered and we can discuss it in that simple way.

The Driving Force (The Muscle)

This is the mechanism that produces the gas flow. This does the work of respiratory muscles or takes over the "the work of breathing".

The Controls Mechanism (The Brain)

- This does the work of respiratory center. This commands the muscles (the driving force—the mechanism that produces gas flow) to do what is required for the patient, as set by the physician. (Rate, Rhythm, Tidal volume. Inspiratory pressure, etc).

A resuscitator bag is a very simple form of ventilator. The driving force or the muscle is the muscles of the operator's hands. The brain or control mechanism is the operator's brain that decides about the phases, volume, etc.

A basic ventilator must do the following:

- Split the **minute volume** calculated for the particular patient based on the clinical assessment, into the required number of **tidal breaths.**
- **As set by the operator provide the following:**
  - The rhythm – I : E ratio
  - Respiratory rate
  - Inspiratory pressure.

East Radcliff Ventilator was a basic ventilator introduced in 1956 which is truly a mechanical ventilator as it functions simply on mechanical principles. Yet it was proved to be a very useful equipment, has been described in the previous chapter (Fig. 7.6).

Now all the possible sophistications have been added to the basic ventilation because of the developments in technology. The modern ventilators have all the different capabilities, modes, and alarms, etc.

Most of the modern ventilators are microprocessor controlled with excellent functional characteristics and are very user friendly with the necessary alarms.

However, we must accept and remember one fact. The sophistication of the ventilator is less important, but the intelligent use of the ventilator by the physician makes a great difference in the ultimate out come of the therapy.

"Unfortunately, many newer modes have been introduced merely on the basis of technical ability rather than as a result of a defined clinical need or demonstrable advantage to the patient."

—J Denis Edwards.

AN IDEAL VENTILATOR THERAPY

An ideal ventilator therapy in any patient requiring mechanical ventilation will results in the following.

- Makes the patient comfortable.
- Improves the chest expansion.
- Improves the oxygen saturation and optimizing CO_2 elimination.
- Normalizes the blood gas level earlier.
- Provides conditions for early weaning.

Ideal Initial Setting

The following settings are advised as initial settings though, smaller variations in one or two parameters are preferred by some.

- Tidal volume: **10 to 12 ml/kg**
- Respiratory rate: **10 to 12/min**
- Peak inspiratory flow rate: **60 L/min**
- I : E Ratio: **1 : 2**
- F_IO_2: **0.5**
- PEEP: **3 to 5 cm H_2O**
- Wave form: **Decelerating Ramp**
- Trigger sensitivity: **1 to 2 cm H_2O**

Here again it is necessary to analyse why the particular initial settings are preferred and what will happen if an alteration is done to a higher or lower setting. It will be ideal to *analyse all the parameters one by one.*

Tidal Volume

- **10 to 12 ml/kg.**
  This is considered ideal for a patient with normal lungs.

- **5 to 7 ml/kg.**
  This lower tidal volume will causes micro atelectasis with resultant hypoxemia.
- Mechanical ventilator tidal volumes are larger than spontaneous tidal volumes **(5 to 8 ml/kg)** to prevent progressive alveolar collapse.
- Atelectasis will develop if a patient breaths at a normal tidal volume without intermittent deep sighs. This problem was averted by intermittent application of sighs about one and a half the tidal volume in olden days. Now the same goal is reached by constant application of larger than normal tidal volume for ventilation.
- *Smaller tidal* volumes are used in situations where the lung is already hyper inflated, as in severe broncho-spasm or in cases with decreased compliance.
- In patients with decreased compliance, large tidal volume may result in an increase in peak inspiratory pressure (PIP) and may cause barotrauma.
- In diffuse diseases such as ARDS, large tidal volumes are *maldisrtibuted to the areas of normal compliance*. This will lead to the overdistension of the more healthy alveoli and thereby *increase the physiological dead space* and also increase the chances for barotrauma.
- In such circumstances it is advisable to use lower tidal volumes **< 12 ml /kg** with a higher respiratory rate.
- Larger volumes than **12 ml/kg** may cause Barotrauma/ Volutrauma, (Stretch Injury) or CVS Decompensation. (It will be discussed in detail later).

Here, **the compressible volume or compliance volume** of breathing circuits of ventilator is to be considered.

- This is the volume of gases which get compressed during positive pressure inspiration within the breathing circuits. It is related to the compliance of the

tubing and the opposing pressure from the patient's lungs.

- The flexible tubing of the ventilator expands and accommodates some of the gases meant to be delivered to the lung. Thus this volume is lost from the tidal volume.
- This volume is delivered from the ventilator into the breathing circuit, but it does not reach the patient' lungs.
- When the *patient's pulmonary compliance is reduced*, there is greater opposing pressure from the lung which will *compress more gases into the tubing*, humidifier, water trap, and internal circuits of the ventilator because of the expansile nature of the tubing. This volume varies grossly with various types of tubes used in the circuit.
- It has to be remembered that at the end of inspiration, this compressible volume, may flow out of the expiratory limb and will be interpreted by the ventilator as part of expired tidal volume.
- The compressible volume can be reduced to a certain extent by keeping the water level in the humidifier as high a possible and keeping the length of the circuit minimal.

This is not easy to calculate it, but not delivered to the patient. It is roughly estimated that for each centimeter of water (cm H_2O) airway pressure 2 to 4 ml of gas will be compressed in the circuit. Imagine that a tidal volume is set as 450 ml and an expansile volume of 70 ml is lost in the circuit, the remaining volume of 380 ml will be delivered to the patient which means the patient is inadequately ventilated. This expansile volume has to be deducted. All modern ventilators have two display panels which can help to rectify this problem to some extent.

Display Panel in the Ventilator

There are two digital displays on the panel.

- The first one shows **what is set on the machine** by the operator.
- The second one shows **what actually the patient receives**.

It is absolutely essential to monitor whether the set tidal volume is delivered to the patient's lungs. The comparison between the two displays will give us the information about it.

Tidal volume monitoring may be done in any one of the following ways,

- *Wright's Respirometer* attached to the expiratory limb. (not in common use now)
- *Volumeter bellows* known as expired air spirometer in some ventilators.
- *Transducers* in the expiratory port. Modern microprocessor controlled ventilators to measure the tidal volume more accurately by this method *(digital monitors).*
- It is always accurate to measure the expired tidal volume (EV_T).
- In a volume cycled ventilator, the desired tidal volume is set on the ventilator, but it is misleading to believe that the patient's lung will always receive the set tidal volume.
- Some of the set tidal volume may be lost as in the leaks in the ventilator circuits, around the endotracheal tube or as discussed earlier, may be lost in the compressible volume in the ventilator circuit.
- That is why the tidal volume measurement is done in the expiratory limb, measuring the volume of gases coming out of the lung (EV_T).

- If the (EV_T) deviates from the set tidal volume by more than 100 ml, then the search for the cause must be made urgently.

Inspired Concentration of Oxygen (F_IO_2)

- "**F_IO2** Should be as high as necessary and as low as possible"
- *It is the percentage of Oxygen in the inspired air* and 100% is mentioned as 1 and 70% oxygen is referred to as F_IO_2 of 0.7, and 60% oxygen in inspired gas is referred to as 0.6 and so on.
- F_IO_2 is a fraction and it can be mentioned that the patient is on 30% oxygen instead of saying that the patient is on 0.3 F_IO_2.
- When mechanical ventilation is initiated, as a measure of caution, it is always better to keep the F_IO_2 as 0.7 to 1.
- After reviewing the first blood gas value, the F_IO_2 may be decreased to appropriately lower level to achieve the goal of clinically acceptable PaO_2 (> 60 mm Hg) with an F_IO_2 of 0.5 or less to minimize the oxygen toxicity.
- If a F_IO_2 of 0.6 is necessary to maintain oxygenation, then the addition of PEEP may be considered.
- Pulse Oximetry must be used continuously for monitoring oxygenation and titration of F_IO_2.
- If blood gas values are used, *it is ideal to wait for 20 minutes* to take a blood sample *after altering the ventilator setting*.
- Increasing the O_2 concentration must be understood as symptomatic treatment.
- In patients treated with Bleomycin (used in cancer therapy) and Amiadarone (used in cardiac arrhythmia) the lung becomes extremely susceptible for oxygen radical mediated injury.

Respiratory Rate

- **Normal: 10 to 20/min**
- It must be set at a rate which is as nearer to the physiological respiratory rate as possible (**10 to 20/min**)
- Most patients, during the initiation of ventilator support may require full ventilatory support.
- The rate at this time selected based on the tidal volume so that the minute ventilation is (RR × V_T = Minute Volume) sufficient to maintain a normal acid base status.
- As the patient starts participating in the ventilatory work, the ventilator RR may be reduced.
- Frequent changes in the respiratory rate are often required based on the observation of the patient's work of breathing and comfort and also on the assessment of the $PaCO_2$ and pH.
- *Slow rates* may be beneficial to the patients with obstructive pulmonary disease, because, as the rate is decreased, more time is available for expiration, and less air trapping will occur.
- *Faster rates* may be needed for patients with non-compliant lungs who require ventilation with smaller tidal volumes to prevent barotrauma by increased airway pressure.
- It has to be remembered that patients on mechanical ventilation are very commonly overventilated that they go for alkalosis with its associated problems. Hence it is desirable that *the patients set their own rate by using a mode of ventilation that allows patient initiated breaths.*
- Depending on the patient's ability, a guaranteed number of breaths may be supplied while the patient is allowed to breath over the rate. Then the patient will adjust to the rate and $PaCO_2$ levels thus maintain a normal acid base status (Eucapnea).

Flow Rate

- **Normal: 40 to 60 L/Min**
- Flow rate *is the speed* in which the tidal volume is delivered.
- It is measured as liters/minute. Normal value is 40 to 60 L/Min.
- If the peak inspiratory flow rate is low, it is definite that in the available inspiration time the tidal volume cannot be delivered.
- So the inspiratory *flow rate is the chief determinant of the inspiratory time* and thus the I : E ratio
- In general an initial flow rate of **40 to 60 liters/min** will satisfy the patient's inspiratory demand and the tidal volume will be supplied in the time so that the desirable inspiration to expiration ratio (I : E) is maintained.
- *Therefore, the flow rate must be adjusted for each patient based on the required I : E ratio, the tidal volume and the respiratory rate.*
- In other words, tidal volume (V_T) must be delivered in the appropriate and comfortable time and the flow rate must meet or exceed the patient's inspiratory flow demand. Otherwise, the patient will experience "Air hunger", the work of breathing will increase, and patient–ventilator dyssynchrony (fighting the ventilator) will result.
- Higher flow rates (> 60 L/Min) will shorten the inspiratory time thereby lengthening expiratory time (decreased I: E ratio) which may be desirable in patients with chronic obstructive pulmonary disease and air trapping.
- Higher flow rate may have the negative effect of increasing the peak inspiratory pressure (PIP) and adversely affecting the distribution of gases as the flow becomes turbulent.

- Lower inspiratory flow rates (20 to 50 L/Min) will prolong the inspiratory time, improve the distribution of gases, and reduce the PIP as a result of more laminar flow of gases.
- Most of the modern ventilators are capable of providing a flow rate in the range of up to 120 to 180 liters/Min, which will cater the need of all clinical situations.

Flow Patterns

It is the modulations in the flow of gases that the ventilator can make while delivering the gases to the lung during inspiration.

Flow is volume per unit time. For a given tidal volume and for a given I : E ratio, the inspiratory flow can be profiled to differently to meet the varied clinical goals of distribution of gases in the lungs.

Therefore, the flow pattern is a characteristic of a ventilator and it has no contribution from the patient.

So, the flow pattern is a characteristic of a ventilator and it has no contribution from the patient.

- As the peak inspiratory flow rate is important for adequacy of distribution and filling of the alveoli, one another factor which modifies it is the Flow pattern (Flow wave pattern).
- The pattern of inspiratory flow can be modified by the ventilator in such a way that it improves the uniform distribution of gases in the lung which is essential for effective gas exchange.
- In fact it is the pattern with which the tidal volume is delivered into the lungs.
- In volume targeted modes of ventilation, the choice of flow pattern is offered.
- In pressure targeted ventilation, only decelerating flow pattern is used; the idea is to achieve the necessary minute ventilation with as little increase in the airway pressure as possible.

Depending upon the type of delivery of gases (configuration of delivery) during inspiration, there are four flow patterns described as standard patterns.

- They are, **Square, Sinusoidal, Accelerating** (ascending ramp) **and Decelerating** (descending ramp).
- Modern ventilators are capable of generating more flow patterns of any one's imagination, which can usually be a modification of the standard four patterns (It shows the technical capability of the microprocessor rather than a defined clinical requirement).
- A *flow pattern is chosen* on the basis of the patient's disease process and the ability of the particular flow pattern to *promote optimal gas distribution* and affect inspiratory pressures.
- There is some evidence to show that a *Decelerating Flow pattern improves the distribution of gases* in patients with diffuse nonhomogeneous disease where adjacent *alveoli require different inspiratory filling time.* This is known as alveoli with *varying time constants.*
- When there is a necessity for a higher inspiratory flow rate and that causes increase in the PIP (Peak Inspiratory Pressure), adjusting the flow pattern and switching from square to sinusoidal may reduce the PIP.
- *Modification **of flow pattern** in an effort to reduce the PIP should be done only when the physician is available at bedside to evaluate any change in the patient comfort and the desired parameters.*

Square

- Peak flow rate is delivered immediately at the onset of inspiration, maintained throughout the inspiratory phase, and abruptly terminated at the onset of expiration (Fig. 8.2).

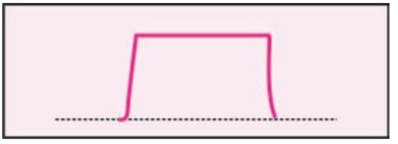


Fig: 8.2: Square flow wave pattern (inspiration)

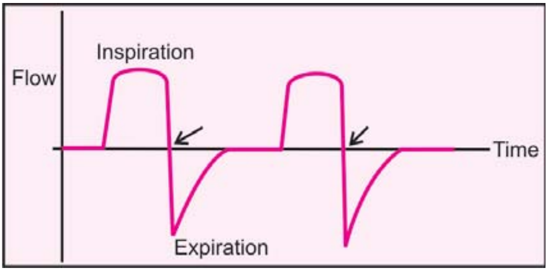


Fig. 8.3: The square wave form showing inspiration and expiration

- ❖ The inspiratory flow remains constant for the entire duration of inspiration.
- ❖ Once the flow stops, the ventilator initiates expiration.
- ❖ The arrow indicates the end of inspiration when the ventilator starts the expiration.

The full square flow wave pattern is shown below with its characteristic (Figs 8.2 and 8.3).

Square Flow Pattern

- The inspiratory flow rises rapidly to a certain level and then held constant until the tidal volume (V_T) is delivered fully.
- Once the entire tidal volume is delivered, the inspiration stops and expiration is started.
- Though theoretically it is said to maintain a constant flow throughout inspiration, in reality there is a tendency for deceleration of flow towards the end of inspiration due the impedance offered by the distending lungs.

- In lungs with increased resistance, this flow pattern is not advantageous as it may increase the peak inspiratory pressure (PIP).

Sinusoidal

- The inspiratory flow rate gradually accelerates to peak flow and then tapers off.
- This is believed to mimic spontaneous inspiratory pattern (Fig. 8.4).
- This pattern may increase the PIP.

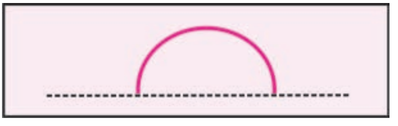


Fig. 8.4: Sinusoidal flow pattern wave (inspiration)

The full sinusoidal flow wave pattern is shown below with its characteristics (Fig. 8.5).

The sinusoidal waveform

- Here the inspiratory flow gradually rises to a peak towards the middle of inspiration and then slowly decreasing to end the inspiration.

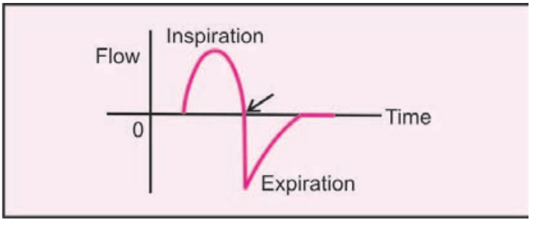


Fig. 8.5: The sinusoidal flow pattern

❖ Note the gradual rise of flow which reaches a peak in the middle of inspiration and gradually falls back to the base at the end of inspiration, when expiration starts.

❖ The arrow indicates the beginning of expiration.

- Though this flow pattern is said to mimic the normal tidal breathing, it has no special advantage for the improvement in the distribution of gases in the lung.

Accelerating (Ascending Ramp)

- Flow gradually accelerates in a linear fashion to set the peak flow rate.
- This pattern is not a commonly used one (Fig. 8.6).

Fig. 8.6: Accelerating flow pattern wave (inspiration)

Decelerating (Descending Ramp)

- This is also a frequently used flow pattern and has many advantages (Fig. 8.7).
- The flow is peak at the onset of inspiration and gradually decelerates throughout the inspiratory phase.
- The flow ceases and the ventilator cycles to expiratory phase when the flow decays to a percentage of peak flow, usually 25% to 10%.
- This pattern may *improve the distribution of gases* when there is inhomogeneity of alveolar ventilation.
- Decreases the dead space, increases the oxygen tension, and reduces the PIP.

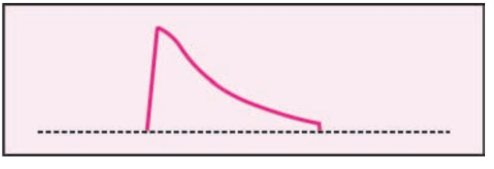


Fig. 8.7: Decelerating flow pattern wave (inspiration)

The full decelerating flow pattern is shown below with its characteristics (Fig. 8.8).

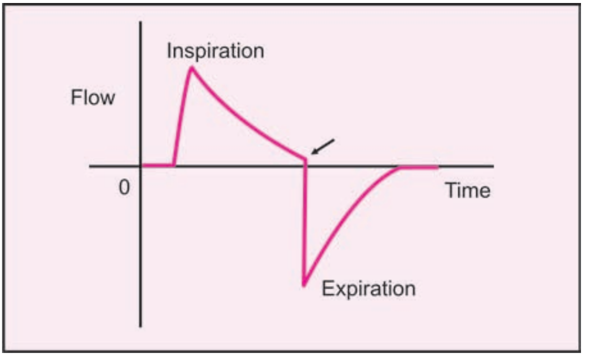


Fig. 8.8: The decelerating flow pattern

❖ The arrow indicates the beginning of expiration.

The Decelerating Flow Pattern

- This is sometimes called as "Reverse ramp pattern"
- The flow of gases rapidly rise to a peak at the onset of inspiration, then the flow rate gradually decreases as the tidal volume is delivered.
- As the lung gets progressively distended during inspiration, the flow required for completing the inflation will be less.
- Therefore, with this flow pattern, lung inflation with the tidal volume is slowed down during the end of inspiration, thus preventing acute rise in airway pressure.
- The advantage of minimising the rise of airway pressure, while delivering the tidal volume is the reason for the preference of this flow pattern.
- The flow pattern generated by a ventilator may be analysed taking decelerating flow *as* an example.

The Figure 8.9 shows all the characteristics of a flow pattern.

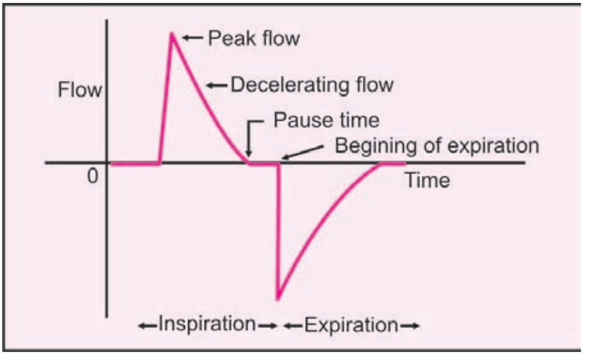


Fig. 8.9: The characteristics of a flow pattern

- Note the peak flow, decelerating curve, inspiratory pause, and beginning of inspiration marked by arrows.
- Note that here is a small pause at the end of inspiration. It may not be present in all the ventilators.

Application of Flow Patterns

The flow pattern is a capability of a ventilator that can modify the distribution of gases and the airway pressure. Therefore, with this fact in mind, it must be looked at to follow the pattern. As an example, the decelerating pattern may be studied.

Decelerating Flow

In this pattern, after having reached an initial high value, the flow falls constantly and gradually. As the filling volume of the lung increases, the pressure also rises and at the end of inspiration the pressure in the lung equals to the pressure in the breathing system, so there is no further flow.

However, usually the ventilator ends the inspiration when the flow falls to a certain percentage of the peak flow to start the expiration (Fig. 8.10).

This flow pattern has some advantages and hence commonly used. The flow is maximal at the beginning and the peak flow is reached quickly. By now, the lungs are filled well with air and the flow gradually decreases (decays) as the filling is complete. When the inspiratory flow decreases to a minimal level, say 25 % of the peak flow (initial flow), the ventilator senses that and ends inspiration. *The expiration is cycled by the ventilator and the flow is directed to the opposite direction and the negative deflection is caused.* The expiratory flow is shown as negative deflection in the flow pattern (Fig. 8.10).

- The decelerating flow wave pattern changes the nature of flow into the lungs and *promotes laminar flow of gases* which is more ideal for uniform distribution of gases.

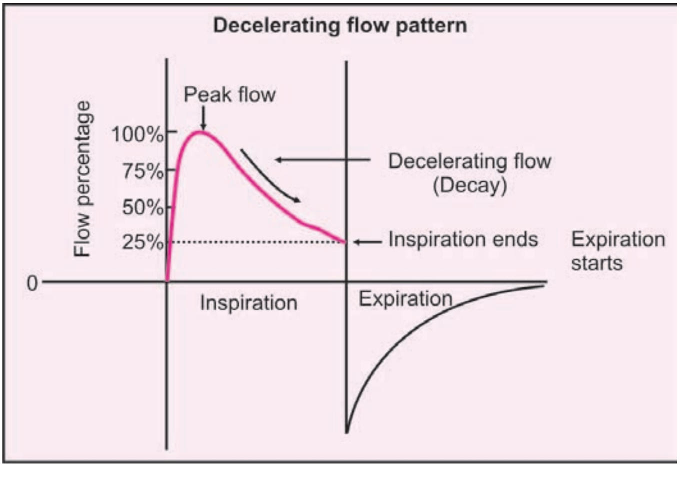


Fig. 8.10: The pattern of decelerating flow

- The laminar flow wave, because of the *parabolic curve* in the flow head (discussed in mechanics of breathing) wedges their way into the airways and alveoli, creating less airway trauma and more uniform gas distribution.
- Decelerating flow-wave has been associated with:
  - Significant reduction in total resistance
  - Improved pulmonary compliance
  - Decrease in dead space ventilation
  - Increase in oxygenation.

Any of the common flow patterns, Square, Sinusoidal, or Accelerating patterns can be set on the ventilator. In fact the newer ventilators have many patterns to choose from.

I : E Ratio (Inspiratory to Expiratory Ratio)

- **Normal I : E ratio: 1 : 2**
- This is the duration of inspiration in comparison with expiration.
- In general in the ventilators it is set as 1 : 2. This is supposed to mimic the normal respiration when the lung functions are normal.
- This means 33% of the respiratory cycle is spent in inspiration and 66% in the expiration. This is generally accepted.
- Shorter inspiratory time contributes to the dead space ventilation by overdistending the most compliant alveoli.
- Longer inspiratory time increases the mean airway pressure, which may lead to hemodynamic instability.
- A I : E ratio of 1 : 3 or 1 : 4 may be used in ventilation of patients with COPD and air trapping, as the longer expiratory time promotes more complete emptying of alveoli and air trapping is reduced.

Clinical Significance

- Correct setting of the I : E ratio is important, because the longer the inspiratory time, the higher the mean airway pressure (MAP).
- When the MAP increases, the chance for **hemodynamic compromise is more.**
- Positive pressure ventilation may reduce the cardiac output.
- It decreases the venous return by abolishing the intrathoracic negative pressure during inspiration and also by applying positive pressure on great veins and preventing them from filling well.
- It increases the right ventricular (RV) afterload and reducing the RV systolic emptying.
- Left ventricular compliance (for filling) is reduced because of the shift of the interventricular septum into the left ventricle by the increased RV end-diastolic volume.

Inverse I : E Ratio

- Normal inspiratory to expiratory ratio is 1 : 2. If the inspiratory time is longer then it is known as inverse ratio.
- So, I : E ratios of 1 : 1, 2 : 1, 3 : 1, 4 : 1 are all inverse ratios.
- Their use is restricted to specific noncompliant lung conditions with nonhomogeneous distribution.
- An inverse I : E ratio is used to improve oxygenation.
- In noncompliant lung the short inspiratory time of a normal I : E ratio allows the unstable alveoli to collapse during the relatively long expiratory phase.
- The inspiratory effect of inverse I : E ratio is to allow the *unstable lung units more time to fill and an equilibration of volume between the alveoli as a result of collateral ventilation.*

- Both the dead space ventilation and the percentage of shunt decrease, because gas is more evenly distributed in the lung.
- The relatively compliant alveoli are not overdistended by gases that preferentially flow to them as in the case of normal inspiration time.
- The simultaneous shortening of expiratory time *prevents the unstable alveoli from collapsing, as the next inspiration starts before they reach the closing volume.*
- Because of the prolonged inspiratory time, there is an increase in mean airway pressure (MAP).
- The increase in MAP is the key determinant for oxygenations as it:
  - Increases the alveolar stability.
  - Increases the recuitment of alveoli.
  - Increases the Functional residual capacity (FRC) (Fig. 8.11).

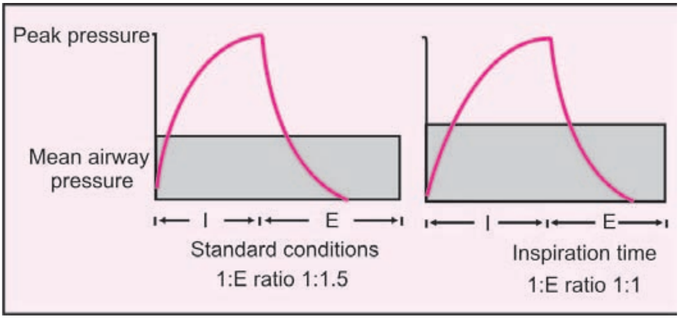
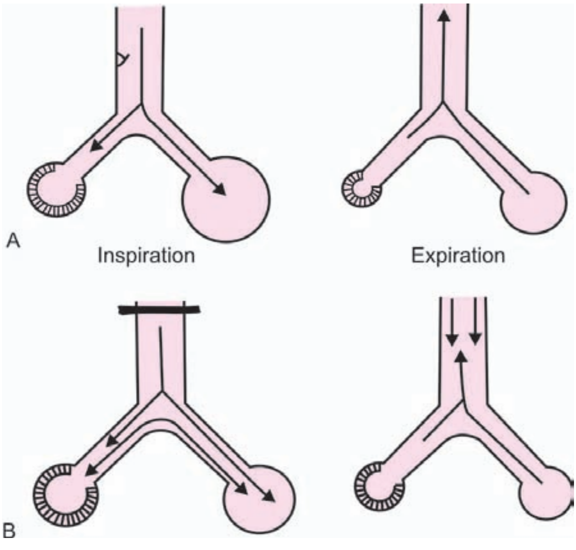


Fig. 8.11: Effect of altering the I : E ratio

❖ As the inspiratory time is increased, the duration for which the positive pressure applied to the thorax also increased.

❖ The mean airway pressure (MAP) rises and increases alveolar stability and recruitment.

However, the increased MAP has negative effects of more positive pressure in the thorax, leading to hemodynamic instability and compromise.



Figs 8.12A and B: Inspiratory and expiratory effects on the distribution of gases
In lung units with varying time constants
In standard ventilation – A,
In inverse ratio ventilation – B

- With short inspiratory and longer expiratory time, noncompliant alveoli do not have time to fill during inspiration. (This forms a shunt) whereas adjacent, compliant alveoli may become overdistended (this forms a dead space).
- On expiration, unstable alveoli collapse as they reach closing volume before the end of the expiratory phase.
- Prolonging the inspiratory time allows noncompliant alveoli more time to fill and equilibration of gases between lung units of varying time constants.
- Reinflation after a shortened expiration causes gases to be trapped in the lung, which creates a PEEP –like effect promoting alveolar recruitment and stability.

- **Intrinsic PEEP or Auto-PEEP:** This is a status which occurs with the inverse ratio ventilation. Because the expiratory time is shortened and the alveoli are not allowed to empty completely on expiration, air gets trapped in the lung (Figs 8.12 A and B). This trapped air creates a pressure in the alveoli that is known as auto-PEEP.

Trigger Sensitivity (Triggering Effort)

- Normal setting: **1 to 2 cm H_2O**.
- The sensitivity is the term used to denote the patient's inspiratory efforts (triggering inspiration) which is used as a trigger for initiating a mechanical breath.
- All modern ventilators have a trigger variable which is manipulated to begin the delivery of inspiratory flow.
- There are two types of triggering available; namely **Pressure triggering** and **Flow triggering.**

Pressure Triggering

- Pressure triggering is the one which is in wide use where as the *Flow triggering is relatively a new concept and is not available in all ventilators.*
- In patient initiated mechanical breaths (assisted breaths), the demand for flow is indicated when the pressure drop in the ventilator circuit is sensed.
- In other words, the sensitivity setting reflects the amount of pressure drop below the base line pressure that the patient must generate in the ventilator circuit, on inspiration, to initiate the flow of gases.
- Generally the sensitivity setting is 2 cm of H_2O less than the end expiratory pressure. Normally it is – 2 cm H_2O.
- If PEEP is used at 5 cm of H_2O, then the inspiratory flow begins when the pressure in the circuit drops to 3 cm H_2O.

- The basic principle is; *the sensitivity must be set in such a way to allow the patient to trigger the ventilator easily.*
- If the patient has to put great effort to initiate the flow of gases or if there is a delay from the time of patient's efforts to the start of the gas flow, then the inspiratory muscle work is increased.

Flow Triggering

- Flow triggering is also known as *Flow By* (FB).
- In pressure triggering the patient has to do some work to initiate the inspiration (efforts). The patient has to create a negative pressure in the ventilator circuit and continue that work for sometime, say a few milliseconds known as the lag time.
- This time lag is because, the patient has to make a inspiratory effort (triggering) and the ventilator assist mechanism must sense this effort and signal the demand valve to open to for allowing the flow of fresh gases.
- In flow triggering the work associated with the patient triggering and lag time is eliminated.
- There is *a base expiratory flow continuously maintained* and monitored by the ventilator's microprocessor.
- Flow sensitivity is an operator chosen *setting that represents how much of expiratory flow has to be decreased by the patient* to trigger the ventilator to deliver the fresh gas.
- When the patient inspires, *the base expiratory flow decreases* which diverts the flow from the expiratory circuit to the patient's lungs.
- This change in the base expiratory flow is sensed by the ventilator to start the inspiration.

Clinical Importance of Sensitivity

- If the trigger sensitivity is set **very low**, then patient must generate more work to trigger the flow of gas.
- Patient may effectively become locked out from initiating gas flow.
- If the setting is **too high**, it may lead to auto-cycling of the ventilator.
- Patient/ventilator dyssynchrony will be caused which leads to fighting the ventilator.

Monitoring

- Expired Air Tidal Volume (discussed earlier)
- Airway pressure
- F_IO_2
- $ETCO_2$
- Disconnection alarm
- Pulse Oximetry
- Blood gas study.

Positive End Expiratory Pressure (PEEP)

Positive end expiratory pressure (PEEP) is the term used wherever a ventilator is in use. As it has been seen earlier while discussing the four phases of ventilation, this is *one of the modifications added to the expiratory phase* with an idea of improving the quality of ventilation and oxygenation.

This is not a primary mode of ventilation, but a method for improving oxygenation by increasing the functional residual capacity (FRC). That is the reason why this is included in this chapter and discussed.

As it is clear that expiration in positive pressure ventilators occurs passively because of the elastic recoil forces of the lung, expiration may be allowed occur to the

baseline pressure zero (atmospheric) or to a controlled positive end expiratory pressure. Both theses features are shown in Figure 8.13.

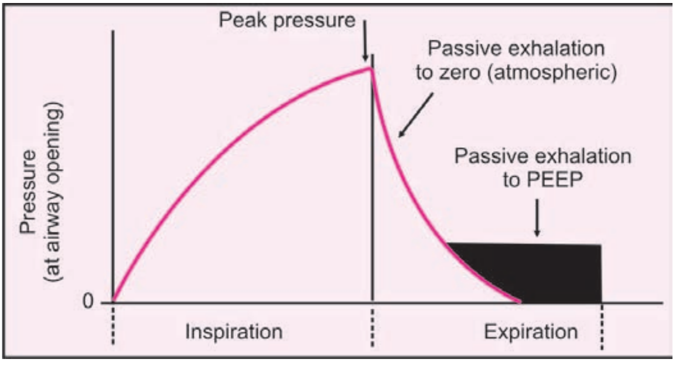


Fig. 8.13: The pressure graph showing passive expiration to zero or PEEP

Positive end expiratory pressure is the application of a constant positive pressure in the airways so that, at the end of expiration, the pressure is never allowed to return to atmospheric pressure. It is mentioned in cm H_2O. The pressure wave is shown in Figure 8.14.

- This positive end expiratory pressure can be applied on a patient who is breathing spontaneously without any other support, for increasing the FRC. Here it is called as Continuous Positive Airway Pressure (CPAP) (Fig. 8.15).
- The positive pressure is actually applied throughout the respiratory cycle, *but it is used for its physiological effects at the end of expiration.*
- The beneficial effects by exerting positive pressure at the end of expiration (PEEP).

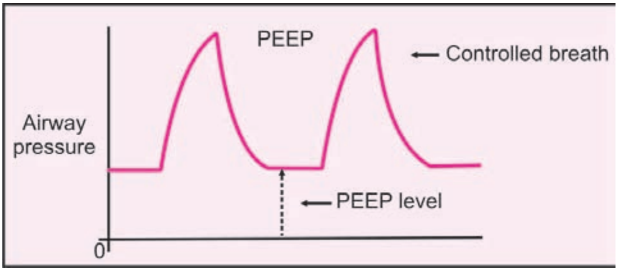


Fig. 8.14: Positive end expiratory pressure applied with CMV

- ❖ The patient is on controlled mechanical ventilation on which PEEP is applied.
- ❖ Note that the end expiratory pressure does not touch Zero (atmospheric pressure).

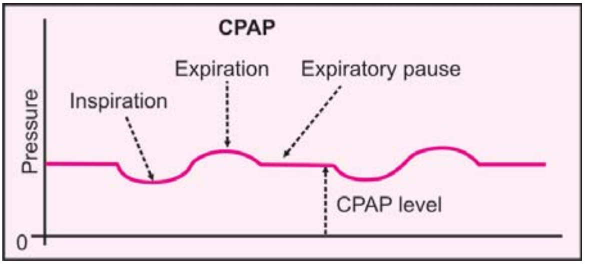
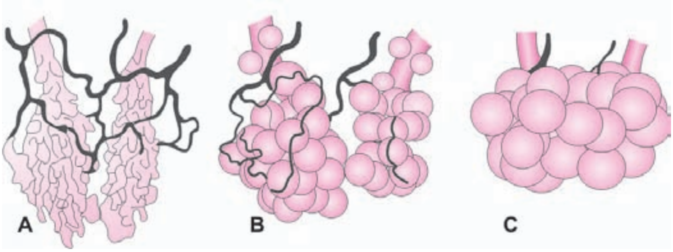


Fig. 8.15: Continuous positive airway pressure (CPAP)

- ❖ Patient is breathing spontaneously as seen in the pressure wave.
- ❖ The end of expiration is not reaching zero (atmospheric pressure).
- ❖ Actually patient is breathing on a CPAP.

- – Recruits atelectatic alveoli (opens up and keep them opened) (Figs 8.16 A to C).
- – Internally splints and distends already patent alveoli.
- – Counteracts alveolar and small airway closure during expiration.
- – Redistributes lung water.



Figs 8.16A to C: The effect of PEEP on alveoli (Recruiting collapsed alveoli)

A. Atelectatic alveoli before PEEP.
B. Optimal PEEP has reinflated alveoli to normal volume.
C. Excess PEEP overdistends the alveoli and compresses the adjacent pulmonary capillaries, creating dead space resulting in hypercapnea.

- PEEP redistributes the extravascular lung water from the alveoli to the perivascular space, where the impact of excess lung water on gas exchange is decreased.
- All these changes due to PEEP cause the following effects:
  - Decreases the intrapulmonary shunting.
  - Increases the functional residual capacity (FRC).
  - Improves lung compliance.
  - Decreases the diffusion distance for oxygen and improves oxygenation.
- For the initial ventilator setting, Keeping $F_IO_2 < 0.6$
- A range from 5 cm H_2O to 15 cm H_2O is normally used. Very rarely 20 cm H_2O is reached.
- For the initial ventilator setting, with a $F_IO_2 < 0.6$, usually 5 cm H_2O is used.
- The useful effects of PEEP are exhausted above 15 cm H_2O.

Indications for PEEP

- Primary indication is to prevent atelectasis in bed ridden patients and sometimes in postoperative patients.

- As a treatment for established atelectasis for reversing it.
- If a patient has PaO_2 of 60 mm Hg or less (oxygen saturation of < 90%) on an F_IO_2 of 0.5 or greater, PEEP is therapeutically indicated for improving oxygenation.
- By addition of PEEP it will be possible to provide better oxygenation with a lower F_IO_2 thereby the chances for pulmonary oxygen toxicity is reduced.
- Critically ill patients are nursed and ventilated usually in supine position and FRC is likely to decrease in this position.
- To preserve a more normal FRC, 5 cm of PEEP may be applied, which will improve oxygenation.
- It must be remembered that the benefits of PEEP will be exhausted possibly after the level of 15 cm H_2O. Hence it is better not to apply very high PEEP.

Contraindications for PEEP

- PEEP will not correct the underlying problems such as *congestive heart failure*, *fluid overload*, *pneumonitis*, *etc*. it only supports oxygenation till the underlying pathology is corrected.
- It is contraindicated in patients with *unilateral lung disease*, because application of PEEP may cause alveolar overdistension in healthy lung, which increases the dead space and redistributes perfusion to diseased lung.
- COPD patients have already increased FRC because of air trapping. PEEP may not improve oxygenation in these patients, but may subject them for the risk of barotrauma and decreases cardiac output.
- Other relative contraindications are; hypovolemia, intracardiac shunts, increased intracranial pressure.

Continuous Positive Airway Pressure (CPAP)

- When a positive end expiratory pressure is applied to a spontaneously breathing patient, with or without the use of a ventilator, who is receiving no other ventilatory assistance, it is called as CPAP.
- Here again the purpose is to increase the FRC.
- Though the mechanism of PEEP and CPAP are identical, the terms should not be interchanged.
- The CPAP can be applied with a ventilator or with a CPAP mask.

Problems Associated with the Use of PEEP

- The first and foremost problem with PEEP is the reduction in cardiac output (CO), caused by the increase in intrathoracic pressure. As it is discussed earlier, it is caused by *three distinct factors*.
- First a decrease in venous return occurs because of increase in right atrial transmural pressure. This in turn is because of referred positive pressure from alveoli.
- This increases the right ventricle afterload, which impairs right ventricle emptying.
- The right ventricle end diastolic volume rises and the interventricular septum is shifted into the left ventricle, impairing diastolic filling of left ventricle.
- The management of this problem is of primary importance as it is likely to end up in reduced tissue perfusion, and oxygen delivery resulting in hypoxia.
- First thing is to ensure that the intravascular volume is adequate.
- After ensuring that the volume is made adequate by infusion of suitable fluids, inotropic drugs to improve the performance of heart and vasoactive agents to support the peripheral vasculature to maintain a systemic arterial pressure in optimum level.

The Method of Applying PEEP

- When indicated, PEEP is set as 5 cm H_2O as an initial setting.
- The assessment of improvement in oxygenation is done and if needed the PEEP may be increased in increments of 3 cm H_2O.
- Each time the change is made, evaluation by pulse oximetry or by blood gas estimation is done. *Ideally blood gas value should be obtained after 20 minutes of change in PEEP*
- The goal is to find the "best" PEEP, which is the least amount of PEEP required to obtain an oxygen saturation of > 92%, PaO_2 > 60 mm Hg, and an F_IO_2 < 0.6. With these levels oxygen delivery to tissues will not be impaired.
- The most important fact is, if PEEP decreases the cardiac output (CO) in an uncorrectable way, then PEEP must be lowered to improve cardiac output and F_IO_2 is increased, if necessary even to toxic levels.

Removal of PEEP

- Once the F_IO_2 could be reduced to 0.6 and the patient is hemodynamically stable, then the withdrawal of PEEP may begin.
- PEEP may be reduced in increments of 5 cm H_2O, each time adequacy of oxygenation is evaluated.
- *Each time the reduction in PEEP is done,* the patient's condition is *allowed to stabilise for 6 hours before another decrease is made.*
- If rapid reduction or withdrawal is done, it will result in hypoxemia because of airway closure and the need to reinstate PEEP possibly at a higher level than previously required.

Auto-PEEP (Intrinsic PEEP)

Definition: *Auto-PEEP is defined as spontaneous development of PEEP as a result of insufficient expiratory time.*

- Expiratory time is inadequate when the lung has not reached its resting expiratory volume before the next inspiration begins.
- Inadequate expiratory time causes gases become trapped in the lung.
- These trapped gases create a positive pressure in the thorax.
- Auto-PEEP may be caused by the following reasons.
  - Rapid respiratory rate
  - High minute ventilatory demand
  - Air flow obstruction
  - Inverse I : E ratio ventilation (Fig. 8.17).
- Auto-PEEP will continue to develop until the elastic recoil forces in the lung overcomes the tendency to trap further gases.

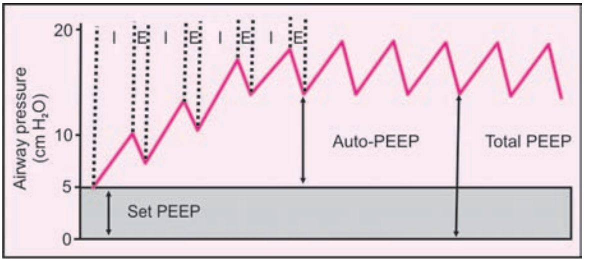


Fig. 8.17: Auto-PEEP developing due to the reduced expiratory time and air trapping

- Auto -PEEP when it occurs unwittingly, it is called by other names such as 'Pulmonary gas trapping', 'Endogenous PEEP', 'Occult PEEP', 'Intrinsic PEEP', Inadvertent PEEP'.

- Extrinsic PEEP is the amount of PEEP that the clinician sets on the ventilator. It is the PEEP we know about and can be read on the display panel at end –expiration.
- Auto-PEEP is the PEEP that we may not know about and cannot be readily detected on the display of the ventilator.
- During expiration the manometer is open to the atmosphere and so reads only the set PEEP.
- For measuring auto-PEEP the exhalation valve must be occluded just before the beginning of next breath. (As an *inspiratory hold* is done for measuring the static compliance).
- Occluding the exhalation port for several seconds allows the ventilator pressure manometer to read both the circuit pressure (set PEEP) and the airway pressure (auto-PEEP). Therefore, the airway pressure manometer reading will reflect the *total* PEEP.
- Now auto-PEEP can be calculated as follows; Auto-PEEP = Total PEEP – Set PEEP.
- The detection and monitoring of auto-PEEP is important because both the PEEP function physiologically in the same manner.
- If it is unintentional, then the clinician who is unaware of its presence will be unable to manage the potentially adverse effects on the patient.
- The adverse effects may be; barotrauma and hemodynamic compromise, it will increase the work of breathing in patient triggered ventilation.

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CHAPTER 9

Available Modes of Ventilation

- *Available modes*
- *Primary modes*
- *Settings on the primary modes*
- *Special modes*
- *Non-conventional modes*
- *High frequency ventilation*
- *New generation*

Originally, the method by which a ventilator *accomplishes the respiratory cycle* is called as a **'Mode'** of ventilation. In other words, it must have all the four phases of the respiratory cycle namely, Inspiratory phase, Change over from inspiration to expiration, expiratory phase, and Initiation of Inspiration (Change over from expiration to inspiration). The modes that have all the four phases are known as **"Standard Modes"** (Eg: Control mode, Assist/ Control mode). There are various newer techniques which have different characteristics and are applied in special situations may be called **"Special Modes".**

After the advent of innumerable ways to support the ventilation, it is better defined as ***"the method or technique by which the patient and the ventilator interact to perform the ventilatory cycle".***

There is always some confusion in the terminology used in ventilator therapy as some authors and manufacturers use different terms to note the same mode.

More over, some authors name every modification done in as phase of respiratory cycle as a 'Mode'. This also leads to further confusion. For example: PEEP is a modification employed in the expiratory phase with an intention of enhancing the oxygenation. A patient can be ventilated by Controlled mechanical ventilation (CMV) with the addition of PEEP on it. Because PEEP by itself is not a primary mode

of ventilation, it can be used only on a primary mode such as CMV mode.

There are only limited numbers of primary modes of ventilation which can be applied individually for ventilating the patient with or without the addition of a setting such as PEEP.

What is the best term that can be used to refer any other modifications applied to the existing primary mode? Can they be called as "Additional Modes", "Special Modes", or 'Settings'? It can be discussed further.

Whatever term by which they are called, one must understand **three main aspects** about these modes.

1. Whether it can *accomplish ventilation independently* or not?
2. When applied, what benefits it can offer to the patient (*advantages*)?
3. What possible adverse effects it can cause during ventilation (*disadvantages*)?

The list given below shows all the available modes of ventilation. The difference whether it is an independent mode or an addition to the primary mode can be well correlated with the Figure 9.1.

Though it is not an exhaustive list of modes, it gives the commonly used modes. Again some of them are no longer in use, still discussed to make it clear how the betterment of it was invented, for example ZEEP was in use before the introduction of PEEP. Similarly NEEP once in use has been abandoned.

1. Controlled Mechanical Ventilation:	**CMV**
2. Assisted Mode Ventilation:	**Assist Mode**
3. Assist/Control Ventilation:	**A/C Mode**
4. Synchronised Intermittent Mandatory Ventilation:	**SIMV**
5. Pressure Support Ventilation:	**PSV**
6. Pressure Controlled Ventilation:	**PCV**

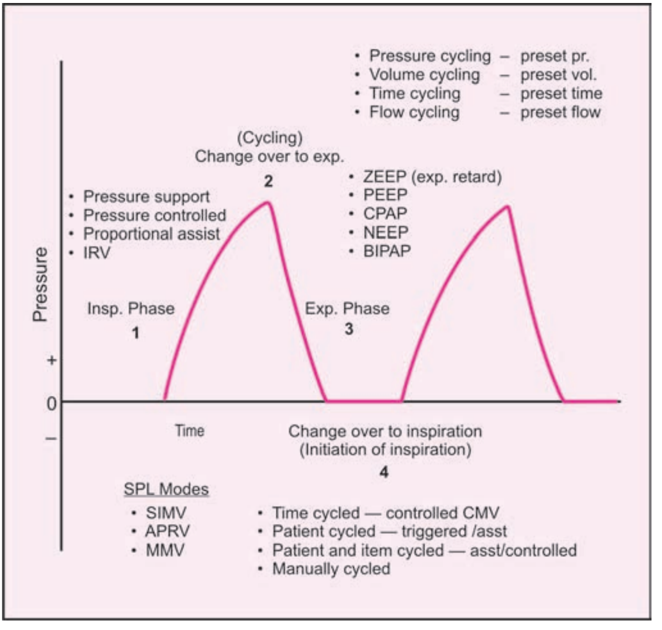

Fig. 9.1: The four phases of respiratory cycle in controlled mechanical ventilation

- ❖ The possible modifications in the inspiratory and expiratory phase are shown.
- ❖ The various types of cycling (changeover from inspiration to expiration are noted at the top.
- ❖ The various types of initiation of inspiration are shown.
- ❖ The Special modes are listed.

7. Inverse Ratio Ventilation: **IRV**
8. Continuous Positive Airway Pressure: **CPAP**
9. Positive End Expiratory Pressure: **PEEP**
10. Zero End Expiratory Pressure: **ZEEP**
11. Negative End Expiratory Pressure: **NEEP**
12. Airway Pressure Release Ventilation: **APRV**
13. Biphasic Positive Airway Pressure: **BIPAP**

14. Minimum Mandatory Volume: **MMV**
15. Proportional Assist Ventilation: **PAV**

In the process of discussion of all these modes, certain degree of repetition of facts becomes inevitable to make things clear some times the figures also. However, it is attempted to keep the repetition as minimum as possible.

If it is carefully looked at the modes given in the list, only the first three are the primary modes of ventilation which can be used independently on a patient. More precisely there are only two primary modes and the third one "Assist/Control" is a combination of the first two modes.

1. Controlled Mechanical Ventilation: **CMV**
2. Assisted Mode Ventilation: **Assist**
3. Assist/Control Ventilation: **A/C Mode**

Four out of the others in the list are only modification in the inspiratory phase namely,

1. Pressure Support Ventilation: **PSV**
2. Pressure Controlled Ventilation: **PCV**
3. Inverse Ratio Ventilation: **IRV**
4. Proportional Assist Ventilation: **PAV**

Another four in the list are only modification in the Expiratory Phase. They are,

1. Zero End Expiratory Pressure: (Expiratory retard) **ZEEP**
2. Negative End Expiratory Pressure: **NEEP**
3. Positive End Expiratory Pressure: **PEEP**
4. Continuous Positive Airway Pressure: **CPAP**

ZEEP and NEEP are no longer in use

The other four modes are again not primary modes. They are named as "Special modes of Ventilation".

1. Synchronised Intermittent Mandatory Ventilation: SIMV
2. Biphasic Positive Airway Pressure: **BIPAP**
3. Airway Pressure Release Ventilation: **APRV**
4. Minimum Mandatory Volume: **MMV**

In this chapter it is aimed to discuss about each mode in detail. In that process of discussion, four aspects are looked into:

- Mechanism by which the mode functions
- Advantages of the mode
- Disadvantages of the mode
- Monitoring the patient which includes,
  - Ventilatory settings
  - Patient data;
    - Peak inspiratory pressure (PIP)
    - Exhaled tidal volume (EV_T)
    - Minute ventilation (V_E)
- **Volume-cycled modes deliver a fixed volume at variable pressure (adults)**
- **Pressure-cycled modes deliver a fixed pressure at variable volume (neonates).**

Mode of ventilation is classified by the mechanism that begins the inspiration.

AVAILABLE MODES

There are two basic categories of modes: **Controlled** or **Assisted.**

Controlled Ventilation

In controlled ventilation, the ventilator initiates the breath and performs all the work of breathing.

Assisted Ventilation

In assisted ventilation, the patient initiates and terminates all or some of the breaths, with the ventilator giving variable amount of support throughout the respiratory cycle.

THE CHOICE

The choice depends upon the patient's efforts for respiration. The mode chosen for the particular patient will depend on, how much of work of breathing the patient can perform. In other words, how much of work of breathing, it is desirable for the patient to perform, considering his pathology.

Breath types are classified based on the following facts,

- Whether the *patient* triggers or *ventilator* triggers the breath.
- How the patient and ventilator *interact* to perform the work of breathing (WOB).

Simply,

- The machine cycled breath may be *mandatory* (fully supported) or *assisted*.
- The patient cycled breath may be *supported* or *spontaneous.*

There are four different types of breaths that a patient using a ventilator may demonstrate in clinical setup. Here the term **cycling** is used to refer to **the initiation of respiration.**

Breath type	*Description*
Machine-cycled **mandatory** breath:	A breath that is triggered by the machine and the machine performs all the work of breathing throughout the cycle. (Eg: CMV)
Patient-cycled **assisted** breath:	A breath triggered by the patient, but the machine performs the rest of the work of breathing (Assist).
Patient-cycled **supported** breath:	A breath that is triggered by the patient, but the patient and the machine interact to perform the work of breathing throughout the remaining phases of ventilation (Assist/Control).

Patient-cycled **spontaneous** breath:	A breath that is triggered by the patient and the patient then performs all the work of breathing. (CPAP on spontaneous breathing)

There are two more terms used in the ventilatory support for a patient. Based on the type of support for ventilation ventilatory support is classified into two. They are, *Full ventilatory support (FVS)* and *Partial ventilatory support (PVS).*

Full Ventilatory Support (FVS)

FSV constitutes mechanical ventilation in which the ventilator performs all the work of breathing (WOB) and must be adjusted to maintain CO_2 homeostasis without any contribution from the patient.

FSV does not mean that the ventilator has complete control of ventilatory process, but it means that the ventilator provides all ventilatory needs of the patient.

Indications

- Initial application of mechanical ventilation required by patients with acute respiratory failure.
- It may be necessary for the first 24 to 48 hours to relieve the patients of fatigue and allow the diaphragm and ventilatory muscles to recover from fatigue and allow the underlying pulmonary pathology to resolve.
- In patients with apnea, those who are heavily sedated, paralysed, drug overdosage, cerebrovascular accidents, and in flail chest where normal negative respiratory effort is detrimental.
- Patients with depressed neurological status such as head injury, where therapeutic controlled hyperventilation is usually employed to counteract cerebral edema.

Partial Ventilatory Support (PVS)

PVS is said to be the situation, when both the ventilator and the patient contribute towards the WOB and maintaining CO_2 homeostasis.

- The main advantage of PVS is allowing the patient to respond to the increase in CO_2 thereby increasing the minute ventilation (V_E) and promoting the use of the respiratory muscles, thus preventing disuse atrophy.
- PVS can be provided by all the common modes by titrating the amount of support offered. The exceptions are those modes that control ventilation such as Controlled mechanical ventilation (CMV) and pure pressure control (PC) ventilation.
- When PVS is used, as the patient becomes better and is able to generate more of the WOB, corresponding reduction is made in the amount of assistance by the machine.
- The *work load must be balanced* between the patient and the ventilator in such a way that it prevents *respiratory muscle atrophy* or *fatigue*.

Which is the "best" mode or the "right" mode for a particular patient in ventilatory failure?

It is a common question in the mind of a beginner. As a matter of fact, it is not very easy to answer. One has to consider and appreciate all the aspects of WOB namely,

- The normal physiological WOB.
- The WOB imposed by the airway and the ventilator circuitry.
- The pathological WOB imposed by the disease.

A thorough understanding of the patient's condition and how each mode could interact with the patient to perform the work is essential.

No general formula or exact parameters can be given to guide the PVS. That is because every individual patient is different in his own way with all the above mentioned load of work.

Ideally ***clinical judgment*** and ***titration of ventilatory support*** play a key role in giving the optimum ventilation maintaining oxygenation and acid-base homeostasis at the same time preventing respiratory muscle atrophy or fatigue.

MODES OF VENTILATION

Controlled Mechanical Ventilation (CMV)

This is **the first** among the four breath types—Machine-cycled **mandatory** breath:

- In controlled mechanical ventilation, the patient receives *a preset number* of breaths per minute of *a preset tidal volume* (V_T) (Fig. 9.2).
- The tidal volume delivered by the machine is called as Mechanical breath.
- The breaths are delivered in *equal time intervals*.
- The machine takes over all the work of breathing
- Patient's effort will not trigger a mechanical breath.
- The controlled mechanical ventilation may sometimes be called as *Controlled Mandatory Ventilation* to make it clear that every breath is a mandatory V_T.

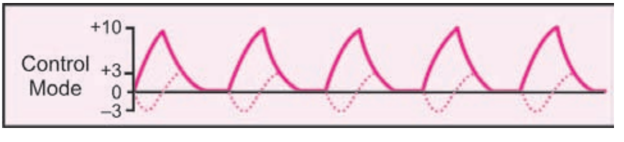

Fig. 9.2: Controlled mechanical ventilation (CMV)

Indications

- Intentional respiratory depression where heavy sedation or neuromuscular block is used.
- When there is minimal or absent respiratory efforts; dysfunction of central nervous system or peripheral nervous system as in encephalopathy, Guillain-Barre syndrome, high level spinal injuries, drug overdosage, etc.
- Flail chest where negative pressure of normal inspiration is detrimental and cause paradoxical moments.
- Used as a fool proof method to ventilate the patient in anesthesia.

Advantages

- In short-term ventilation, it maintains the best form of artificial ventilation as the clinician expects.

Disadvantages

- Before the advent of Assist/Control mode, CMV was extensively used. If the patient is awake and attempts to breathe, he cannot achieve the breath because he cannot enter into the mechanical breaths and is locked out.
- This will increase the work of breathing and also cause a sensation of air hunger.
- This mode is unsuitable for the patient who attempts to breathe, because it may cause patient-ventilator asynchrony (fighting the ventilator).
- A patient who can generate spontaneous respiratory effort must be heavily sedated or paralysed to enhance the patient comfort and ventilator efficiency, if this mode is to be employed.

- That degree of sedation and paralysis are not free from their own inherent problems. So, if the patient's breath is not detrimental to the improvement, some other mode of ventilation must be chosen.
- It has been established that respiratory muscle weakness and disuse atrophy may result if CMV is used for more than 40 hours. This will make the weaning from the ventilator difficult.
- In CMV alveolar ventilation and respiratory contribution to the acid base balance is completely controlled by the clinician. Acid-base balance must be closely monitored and ventilator settings adjusted with changing physiological scenario.

Important Points to Note

- Depending upon the compliance and airway resistance, the peak inspiratory pressure (PIP) will vary in this volume cycled ventilation.
- Exhaled tidal volume (EV_T) must be watched, as the set tidal volume (V_T) may be lost in the circuit. If the EV_T deviates from the set V_T by 100 ml in an adult, we have to look for a source of loss of V_T.
- Patient-ventilator asynchrony may be due to inadequate setting of *flow rate* or *respiratory rate* to meet the patient's ventilator needs.
- Sedation must be adequate in patients with ability to initiate spontaneous breathing.

Assist Mode

This is **the second** among the four breath types discussed in the beginning of this chapter: Patient-cycled **assisted** breath.

A breath is *triggered by the patient*, but the machine *performs the rest of the work* of breathing (Assist).

- This mode delivers a patient triggered tidal volume.
- The tidal volume is preset by the physician.
- Whenever the patient attempts to initiate inspiration, he initiates the ventilator to deliver a breath (Fig. 9.3).
- Each such breath will be of volume that has been set by the physician, *'Mechanical breath'*.
- The patient is free to choose his own respiratory rate and thus in assist mode the *patient determines the rate, and the physician sets the tidal volume*.
- The mechanism of triggering has been discussed in detail in the chapter on basic concepts (Chapter 8).

Disadvantage

This mode has the advantage of allowing patient to take part in his breathing by initiating it. Because there is *no back-up facility in this mode*, in case the patient stops breathing, there is no negative pressure to trigger the ventilator breath. This is a dangerous situation that the patient will become apneic.

Hence this mode alone is no longer in use. It is combined with control mode and is known as Assist/Control mode.

Fig. 9.3: Assist mode ventilation

Assist/Control Mode

This mode forms **the third** type among the four breath types discussed, Patient-cycled **supported** breath. This is

the most commonly used mode for starting ventilator support.

A breath that is *triggered by the patient*, but the *patient and the machine interact to perform the work of breathing* throughout the remaining phases of ventilation (Assist/ Control).

- The ventilator delivers *a preset number of breaths* at a *preset tidal volume* (V_T).
- This preset number of breaths is called as the **"Back up rate".**
- In between these machine initiated breaths, the patient may trigger spontaneous breaths.
- A sensing device in the ventilator, which detects the fall in airway pressure below end expiratory pressure during the inspiratory effort of the patient. Every time the device senses this, the ventilator delivers a breath of preset V_T. This is known as "**patient triggered breath".**
- In this mode all the patient initiated breaths (triggered breaths) are delivered as in assist mode, *provided the patient breathes at a rate that is below the back up rate.*
- If the patient fails to trigger a spontaneous breath within the predetermined cycle period, the patient will get a ventilator breath (Fig. 9.4A).

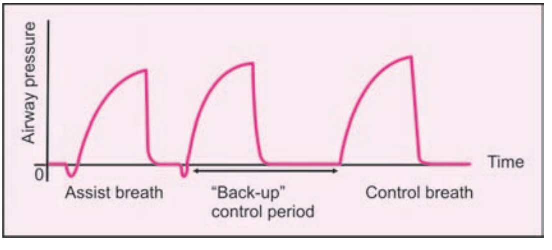

Fig. 9.4A: Assist/Control mode of ventilation

❖ Note that the patient failed to trigger a spontaneous breath, and within the back-up time a control breath is delivered.

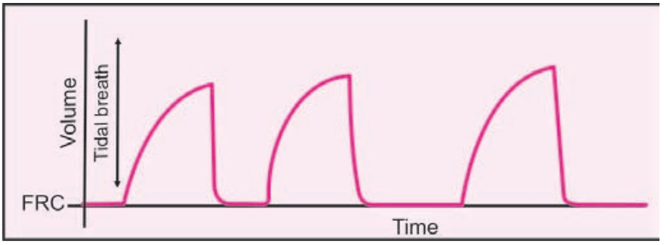

Fig. 9.4B: Volume graph of Assist/Control mode

- If there is any fall in the patient's spontaneous rate of breathing, the machine will deliver breaths at the back up rate with the set tidal volume (Fig. 9.4B).
- Thus this mode *guarantees the basic ventilatory requirements*, at the same time it *dose not prevent the patient from breathing* spontaneously.
- The *patient cannot vary the volume* of the spontaneously initiated breaths.
- The only work the patient has to do is an inspiratory effort to create the negative pressure necessary to trigger a ventilator breath. The ventilator performs the rest of the work of breathing.
- Full ventilatory support is assured, provided the back up rate is set reasonably high enough.
- The patient is relieved of most of the work of breathing and allows rest for the muscles of respiration.
- Though the work of breathing is mainly done by the ventilator, the patient has to spend energy needed to trigger the ventilator.
- The trigger sensitivity has to be set at the minimum for the patient to initiate inspiration with minimal effort.
- If the *trigger sensitivity is set at too high a level*, the amount of energy required to trigger the ventilator can be considerably high, leading to an increased work of breathing.

- ***Pressure-support ventilation (PSV)*** *is a modification of this system; mechanical augmentation of spontaneous breaths up to a preset level of positive airway pressure as long as patient demand exists.* The patient retains the control of the rate (it will be discussed in detail later in this chapter).

Example

- A patient is breathing at a rate of 20 breaths/min.
- If we set a tidal volume of 500 ml and a back-up rate of 10 breaths/minute, the ventilator will deliver 500 ml for every breath triggered by the patient.
- If the rate falls too low, for example 5/min, the back up rate of 10/min will be provided by adding 5 more breaths to that. Here a minute volume of 5 liters is assured.
- If the patient stops breathing, then the tidal volume of 500 ml would be delivered at the back-up rate of 10 breaths/min thus assuring a minute volume of 5 liters.
- *If all the inspiratory attempts of the patient are supported, hyperventilation is inevitable.*

Indications

- Patient has normal respiratory drive, but the muscles are weak to do the work of breathing. For example, recovering from anesthesia.
- Patient has normal respiratory drive, but the respiratory muscles are not able to do the increased work of breathing (WOB) due to lung diseases where lung compliance is decreased, or resistance is increased.
- When it is desirable to allow the patient, to set his own rate, thereby assist in maintaining a normal $PaCO_2$.

Advantages

- Minute ventilation is guaranteed.
- Set tidal volume is guaranteed with each breath.
- The respiratory rate can be dictated by the patient, if he can over-breathe the set rate.
- The triggered breath is in synchrony with the patient's inspiratory effort, making it comfortable.
- Rests the patient and unloads the respiratory muscles.

Disadvantages

- Because of anxiety, pain, etc. patient may hyperventilate, and it will cause respiratory alkalosis.
- Significant respiratory alkalosis may depress the respiratory drive.
- Hyperventilation is likely to cause auto-PEEP, because of shorter expiratory time.
- If the lung mechanics are poor, airway pressure may rise too high.
- Work of breathing may rise if the *trigger sensitivity* and *flow rate* are not set properly.

Important Points to Note and Monitor

- The peak inspiratory pressure (PIP) in this volume cycled ventilation is variable with changes in compliance and resistance.
- Though the T_V is set in the panel, it is not guaranteed that the volume is delivered. The exhaled tidal volume (EV_T) must be monitored.
- If, EV_T deviates from the set V_T by more than 100 ml or more, look for a loss of V_T.
- Patient's subjective feeling of comfort must be looked for.

- The *trigger sensitivity* and *the flow rate* are the two variables that affect the patient's work of breathing in A/C mode.
- Airway pressure manometer must be monitored and the sensitivity is adjusted so as to allow minimal triggering effort.
- Flow rate must be adjusted to meet the inspiratory demand of the patient.
- Close monitoring of acid-base status is necessary. If the patient is hyperventilating, consider sedation or changing to a mode in which patient has greater control. For example: SIMV or pressure support ventilation.

To be more correct, these three modes are the basic modes of ventilation. All the other modes to be discussed hereafter are either known as **special modes** or modifications set on the existing basic mode.

Intermittent Mandatory Ventilation (IMV)

It is **a special mode** of ventilation applied to a patient breathing spontaneously to support the ventilation, *improve the quality* and *quantity* of ventilation. It is a blending of spontaneous breathing with mechanical ventilation.

There is a continuous flow of gases available throughout the respiratory cycle. The patient breathes at his own rate. *The mandatory mechanical breaths of set volume are delivered at the set time on that.*

This mode was originally started in an effort to create a mode in which the patient could interact with the ventilator, at the same time use the respiratory muscles, so that it will be useful in weaning patient from ventilator support. Though this mode is not in common use today, it is discussed here only to know about the problems

associated with its use and how the problems were managed till the better analogue SIMV was introduced. The features are:

- The patient receives *a preset number* of breaths of a *preset tidal volume* (V_T).
- The physician *sets the tidal volume* and determines *how many mandatory breaths* are to be delivered per minute. The mandatory breaths are delivered to the patient intermittently, at regular intervals of time (Fig. 9.5).

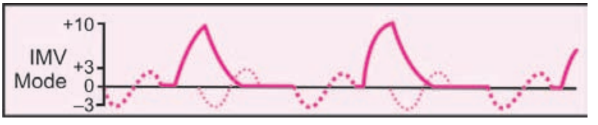

Fig. 9.5: Intermittent mandatory ventilation (IMV)

❖ This figure shows five spontaneous efforts and two mandatory breaths.
❖ The bold dotted lines indicate the patient's spontaneous respiration.
❖ The mandatory breaths are set at regular intervals of time.
❖ It can be noted that the mandatory breaths fall on the expiratory phase of patient's spontaneous breaths. This will cause asynchrony, breath stacking and barotrauma.

- Between these mandatory breaths (compulsory breaths), the patient will be breathing spontaneously at his own desired rate.
- The tidal volumes of the spontaneous breaths depend on the muscular respiratory efforts the patient is able to generate.
- The lower the IMV rate, the more spontaneous breaths the patient will initiate, thereby assuming a greater portion of ventilatory work.
- The difference between the IMV and Assist/Control modes is the volume of patient initiated breaths.
- In Assist/Control mode the assisted breaths and control breaths have a set volume that is guaranteed.

- In IMV the tidal volume of patient initiated breath is variable, but the mandatory breaths are of set volume and constant.
- When the patient is able to generate more work of breathing, the number of mandatory breaths is reduced accordingly.

Indications

- In patients with normal respiratory drive, but the respiratory muscles are not able to do all the work of breathing (WOB)
- In situations where we would like to allow the patient to have their own respiratory rate thereby assist to maintain a normal $PaCO_2$.
- Respiratory muscle conditioning
- For weaning the patient from ventilator support.

Advantages

- Hyperventilation is not a problem as in A/C mode, because the patient can modify rate and volume of ventilation on spontaneous breaths and therefore maintain normal CO_2 levels.
- Respiratory muscular atrophy is relatively less possible as the patient participates more in ventilation using their own muscles to a greater degree when compared to CMV or A/C modes.
- Hemodynamic effects of positive pressure ventilation are less, as ventilation will occur at lower mean airway pressure when spontaneous breaths are taken.

Disadvantages

- The major disadvantage is that the mandatory breath may be *delivered during any phase of patient's spontaneous of ventilatory cycle.*

- It may cause breath stacking, when the mandatory breath falls during or at the end of inspiration.
- The breath stacking will cause patient-ventilator dyssynchrony, discomfort, inadequate ventilation, and potentially barotrauma.
- If breath stacking causes pressure limiting on inspiration, then the ventilator will vent the rest of the V_T to atmosphere.

Important Points to Note and Monitor

- Patient's respiratory rate has to be monitored. If the respiratory rate increases, the tidal volume (V_T) of the spontaneous breaths must be reassessed. It should be 5 to 8 ml/kg.
- If the patient becomes fatigued, the tidal volume will slowly fall and rapid shallow breathing may be caused. Rapid shallow respiratory pattern will lead to atelectasis, reduction in compliance, and further increase in work of breathing (WOB).
- Peak inspiratory pressure (PIP) is monitored, as it is variable in this volume cycled mode depending upon the changes in compliance and resistance.
- Exhaled tidal volume (EV_T) of mandatory breaths is to be monitored. It is not guaranteed that the preset tidal volume is delivered because of factors discussed earlier. If EV_T deviates from the set V_T by 100 ml or more in an adult, search for the source of loss.
- Monitor the volume of spontaneous breaths. It should be 5 to 8 ml/kg. If it is less than 5 ml/kg, it indicates that the patient has less muscle strength to generate inadequate tidal volume. It will lead to atelectasis.
- Patient comfort and patient-ventilator synchrony is observed carefully. If the patient is not comfortable, the sensitivity and the flow rate are adjusted appropriately.

- If patient-ventilator asynchrony is a problem, particularly during weaning period, talk to the patient and gain his confidence and reassure him that he will be taken care. Teach him to work with the ventilator. If patient discomfort remains as a problem, sedation to a level that does not depress the respiratory drive may be used and T-piece trials with intermittent A/C mode may be another option for weaning.

Synchronized Intermittent Mandatory Ventilation

It is seen that in IMV, the major problem is patient-ventilator asynchrony. To *prevent this asynchrony an innovation was designed*, where the ventilator detects the onset of patient's spontaneous inspiratory effort and delivers the mandatory breath synchronizing with the effort. That is Synchronized Intermittent Mandatory Ventilation (SIMV). The difference between IMV and SIMV is shown in Figure 9.8A.

- In SIMV, the patient is guaranteed a preset number of breaths of a preset $V_{T.}$
- Between these mandatory breaths, patient can breathe spontaneously.
- The tidal volume of the spontaneous breaths depends upon the muscular effort the patient can generate.
- Instead of delivering the mandatory breath at a precise time, regardless of where the patient is in the ventilatory cycle as in IMV here, *the ventilator senses the patient's negative inspiratory effort and delivers the mandatory breath synchronizing with patient's inspiration* (Fig. 9.6).
- This is achieved as the ventilator monitors the patient's spontaneous, *negative inspiratory effort* and marks *a window of time* and delivering the mandatory breath within the timing window synchronizing with the patient's inspiration (Fig. 9.7).

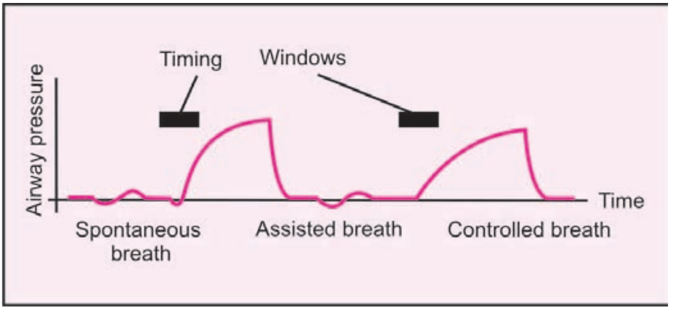

Fig. 9.6: Synchronised intermittent mandatory ventilation (SIMV)

❖ The first mandatory breath is delivered in response to the inspiratory effort.

❖ The second mandatory breath is delivered at the timing window, as there is no spontaneous effort. Ventilator will reset the timing window accordingly.

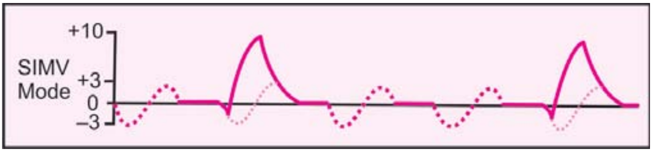

Fig. 9.7: Synchronised intermittent mandatory ventilation (SIMV)

❖ Note that the mandatory breaths fall exactly during patient's inspiratory efforts.

- If the patient does not make an inspiratory attempt, within the timing window, the mandatory breath is delivered at the scheduled time.
- The ventilator then resets the window in response with the next spontaneous inspiratory effort.

The Difference between the SIMV and A/C Modes

- In A/C mode, the V_T of the patient initiated breaths are of guaranteed of preset value.

- In SIMV, the V_T of patient initiated breaths is variable dependent on the patient's effort and lung characteristics.

Indications

- In patients with normal respiratory drive, but the respiratory muscle are unable to do the WOB.
- Conditions where it is desirable to allow the patient to set their own rate, thus assisting in maintaining a normal $PaCO_2$.
- Weaning the patient off the ventilator.

Advantages

- As the mandatory breaths are synchronized with the inspiratory efforts of the patient, better patient comfort is assured.
- Breath stacking is prevented, so the risk of barotrauma is avoided, and loss of tidal volume because of pressure limiting is prevented.
- Hyperventilation is not a problem as in A/C mode.
- Fewer chances for respiratory muscle atrophy, as the patient uses the muscles to a greater extent.
- Haemodynamic compromise is less because the patient ventilates at a lower mean airway pressure (Paw)
- SIMV may be applied for a patient breathing spontaneously with CPAP.

Disadvantages

- The mandatory breath in SIMV is provided through a patient triggered demand flow system. The demand valve opens when the pressure drops in the circuit. The demand flow system may offer considerable resistance which may add to the work of breathing (WOB).

- In newer ventilators this problem is reduced by incorporating better technology.
- This problem may be eliminated by the use of pressure support ventilation.

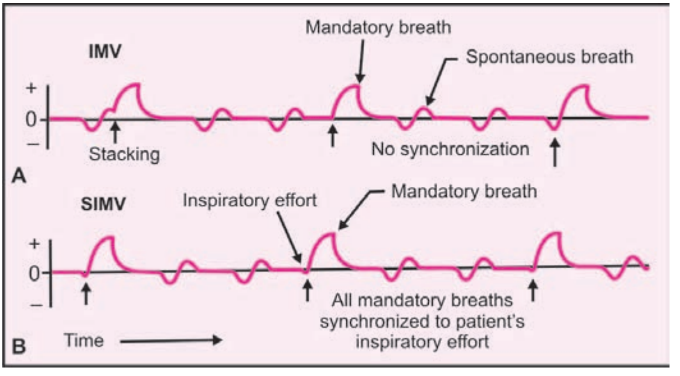

Fig. 9.8A: Comparison of IMV and SIMV

❖ The pressure wave forms of IMV and SIMV.
❖ Mandatory breaths are marked with vertical arrows.
❖ In IMV, the mandatory breaths are given at equal time intervals regardless of where the patient in the ventilatory cycle. It may cause breath stacking.
❖ In SIMV, mandatory breaths are delivered in synchrony with the patient's negative inspiratory effort.

Points to be Noted and Monitored

- Patient's respiratory rate must be monitored. If the respiratory rate increases, the tidal volume of spontaneous breaths must be assessed. The value must be 5 ml to 8 ml/kg.
- If the patient becomes fatigued, then rapid shallow breathing pattern may be caused. This will result in atelectasis, fall in compliance and increased WOB.
- Peak inspiratory pressure (PIP) must be monitored, as it is variable in this volume cycled ventilation. It will vary with changes in compliance and resistance.

- EV_T of mandatory breaths must be monitored to ascertain whether the set volume is delivered. A deviation of 100 ml or more from the set value needs a search for the source of loss.
- Patient comfort must be monitored. If not comfortable, the sensitivity and flow rate must be checked and adjusted.

Now it is worth comparing the modes that have been discussed so far namely, CMV, Assist, IMV, and SIMV. This comparison will give a orientation of the modes concerned (Fig. 9.8B).

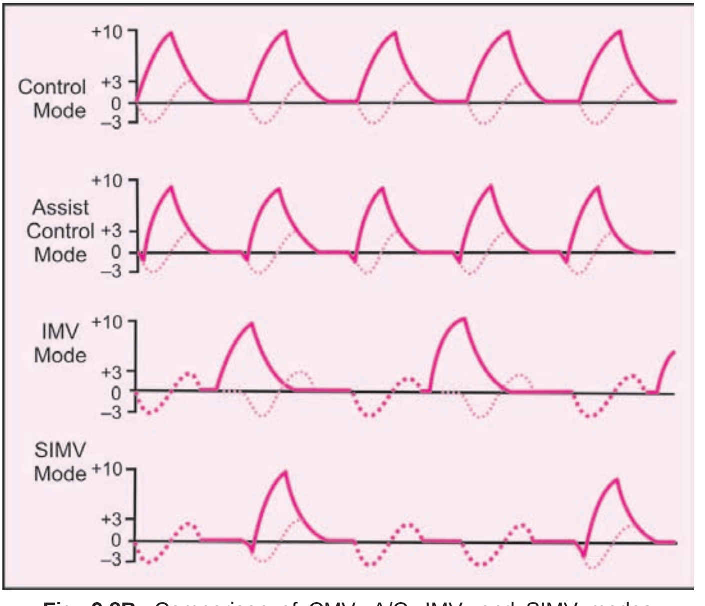

Fig. 9.8B: Comparison of CMV, A/C, IMV, and SIMV modes

- ❖ Thick solid lines represent ventilator breaths.
- ❖ Thick dotted lines represent spontaneous breaths.
- ❖ Thin dotted lines represent the position of normal respiration if present.

The features that could be noted and compared are:

- In control mode (CMV), all the breaths are mechanical breath of uniform time and pressure.
- In Assist/Control mode (A/C), in this tracing, all the breaths are patient initiated mechanical breaths, i.e. the negative deflection is the patient effort which is sensed by the machine and the mechanical breath is delivered. All breaths are of same pressure.
- In IMV mode, two mandatory breaths are delivered on a spontaneous breathing at a fixed time interval without considering the phase of spontaneous respiration.
- In SIMV mode, two mandatory breaths are delivered exactly at the start of inspiration. This synchronization is achieved by the sensor which identifies the inspiratory negative deflection of inspiration.

Continuous Positive Airway Pressure (CPAP)

Definition: This is the positive pressure applied continuously through out the respiratory cycle, to a spontaneously breathing patient.

Though discussed briefly in the previous chapter, the details are given below.

CPAP and PEEP are identical; they are not separate modes of ventilation, as they do not provide any ventilation.

- In CPAP, the patient does all the work of breathing (WOB).
- Therefore, it is essential that the patient has adequate respiratory drive with a reliable tidal volume (V_T), as there is no support or no mandatory breaths are given.
- The main purpose of CPAP is to provide a positive pressure at the end of exhalation, thus preventing alveolar collapse, increasing the functional residual capacity (FRC), and improving oxygenation.

- The CPAP can act *as an internal splint for the unstable alveolar units* and prevents them collapsing. Thus it keeps the unstable alveolar units from collapsing, *recruiting them into useful ventilation.*
- Its useful purpose is at the end of expiration. Instead of giving the positive pressure at the end of expiration, it is applied throughout the cycle; it is called as continuous positive airway pressure (Fig. 9.9A).
- It is identical to PEEP in its physiological effects.
- The term CPAP is used when the baseline pressure is elevated on a patient who is breathing spontaneously and the term PEEP is used when the baseline pressure is elevated in a patient who is on some form of ventilator support (Fig. 9.9B).
- The pressures employed, as in PEEP ranges from 5 cm H_2O to 15 cm H_2O.

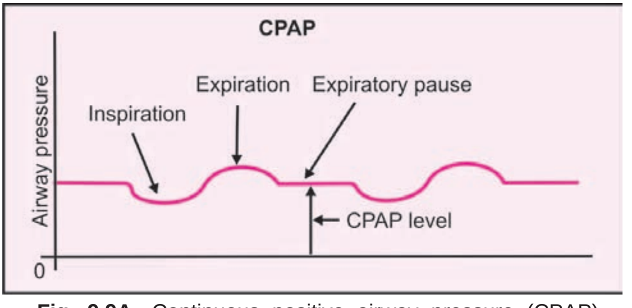

Fig. 9.9A: Continuous positive airway pressure (CPAP)
❖ Note that, the patient breaths spontaneously on a CPAP.
❖ Inspiration, expiration and the expiratory pause could be seen.

Indications

- Ideally used in patients where adequate ventilation with *inadequate oxygenation because of reduced FRC* in diseases like atelectasis and retained secretions.

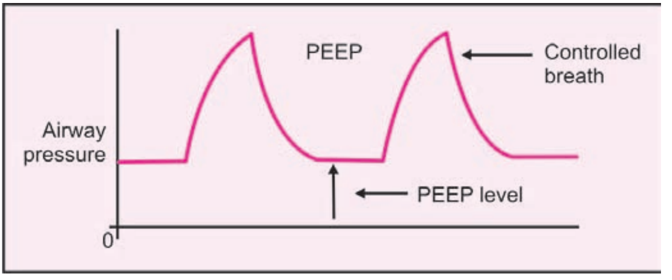

Fig. 9.9B: Positive end expiratory pressure (PEEP)

❖ Note that the patient is given CMV on a set PEEP level.

- For patients where ventilation is adequate, but endotracheal tube is needed for toileting purposes.
- Patients who are weaned from ventilator, a CPAP through a mask will improve the alveolar stability and improve FRC.

Advantages

- The first benefit of CPAP is that it prevents atelectasis and splints smaller airways collapsing by raising FRC.
- The increased FRC improves the oxygenation, as FRC is the ready source of oxygen for diffusion.
- The continuous positive pressure opens up the collapsed alveoli (recruiting the alveoli) which eventually take part in diffusion, thereby improving oxygenation.
- Restoration of ventilation by opening up the alveoli which were so far collapsed (perfused but not ventilated) by recruiting them reverses the hypoxemia.
- The compliance improves because of active participation of additional alveoli and for a given pressure more expansion (change of volume) is possible. Therefore, the WOB decreases.

- CPAP can be used both in intubated and non-intubated patients. Nasal and oro-nasal masks are available for that purpose.
- In the process of weaning the patient from ventilator, because the patient is still connected to the ventilator, the problems of low V_T and apnea are indicated by alarms. Delivering mandatory breaths, in the event of apnea is possible. All these are not possible if T-piece weaning is attempted.

Disadvantages

- Like any other positive pressures, reduces cardiac output because of reduced venous return, increase intracranial pressure, and barotrauma are the possible risks.
- With the commonly employed CPAP of 5 cm to 10 cm H_2O, the adverse effects are uncommon unless there is coexisting hypovolemia or cardiac dysfunction.

Points to be Noted and Monitored

- The respiratory rate should not be more than 25/minute. In case of increased rate, EV_T must be monitored.
- If the patient develops rapid shallow breathing and gets fatigued, it will lead to *atelectasis, reduced compliance, and increased work of breathing* (WOB).
- The EV_T must be 5 ml to 8 ml/kg. If it is less than 5 ml/kg, it indicates that the patient does not have sufficient muscle power to generate adequate V_T. It will promote atelectasis. The patient must be switched over to a mode that gives more support.
- Patient's comfort is checked. If uncomfortable and complains that he does not get enough air, *flow rate* must be adjusted. If patient is anxious, give counseling and sedative to the level of not depressing the ventilation.

Positive End Expiratory Pressure (PEEP)

This has been discussed in detail in Chapter 8 (page 261).

Pressure Support Ventilation (PSV)

Patient determines respiratory rate (RR), minute ventilation (V_E), and inspiratory time. It is purely a spontaneous mode.

- Triggered by patient's own breath
- Cycled by flow
- Limited by pressure
- Affects inspiration only
- This mode is a form of manipulation in the inspiratory phase for supporting the ventilation.
- It is applied in a patient breathing spontaneously to support inspiration.
- It is designed to *augment the V_T of spontaneously breathing patients and over come any increased resistance* from the endotracheal tube, breathing circuit (tubing, connectors, and humidifier).
- The patient's spontaneous inspiratory activity is augmented by the delivery of *a preset amount of inspiratory positive pressure*.
- The main setting in this mode is *inspiratory pressure*. The other ventilator parameters that need to be chosen by the clinician are *triggering sensitivity, PEEP, and the pressure support slope* (Fig. 9.10A).
- The slope indicates the rapidity of pressure rise at the beginning of inspiration (Figs 9.10A and B)
- Just at the onset of inspiration, when the patient triggers the inspiration, a preselected amount of *'pressure support' (PS) is delivered and held constant throughout the inspiration and to promote the flow of gases in the lungs.*
- Here, the tidal volume (V_T) not preset. *The tidal volume is variable* as, it is determined by the patient's effort, the

amount of pressure support (PS) applied, and compliance and resistance of the system (the patient and the ventilator).

- The gas flow is delivered by *a decelerating flow wave pattern*. Therefore, the flow rate naturally diminishes (decays), as the patient's lungs fill with air on inspiration (Figs 9.10A and C).
- The ventilator will end the inspiration when, the peak inspiratory flow rate decreases to a minimal level, say 25% or 5 L/min depending upon the model of ventilator. This pressure support level can be adjusted as per the requirement (Fig. 9.10D).
- Considering this fact, it is seen that the cycling is done by the decrease in the flow.

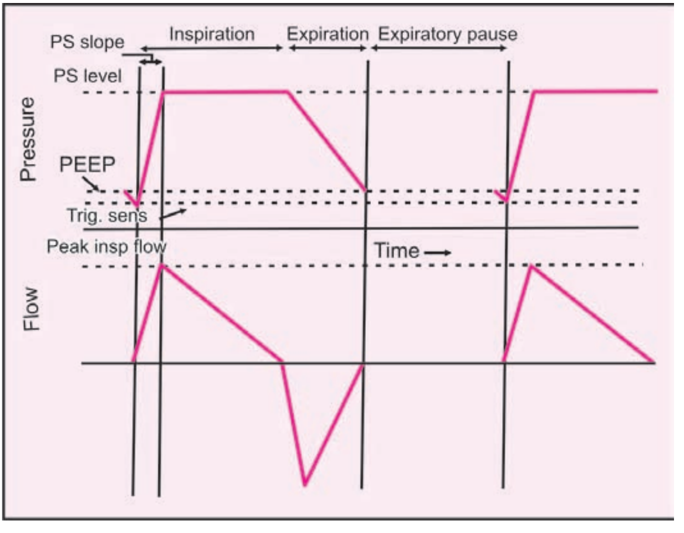

Fig. 9.10A: Pressure support ventilation (PSV)

❖ Note that every breath is patient triggered and the machine gives the pressure support till the patient continues to inspire.

❖ Trigger sensitivity, inspiratory pressure, PEEP, and the pressure support slope are set as needed by the physician.

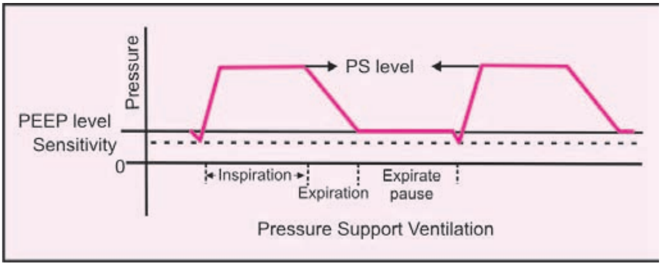

Fig. 9.10B: Pressure waves in pressure support ventilation

- ❖ Note the inspiratory effort of the patient as a small negative deflection at the start of every pressure supported breath.
- ❖ Airway pressure rises rapidly and remains constant in the pressure support level throughout the inspiration.

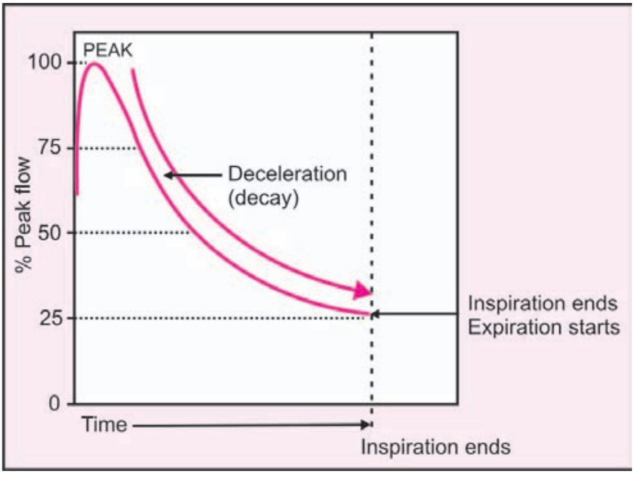

Fig. 9.10C: Decelerating flow pattern in pressure support ventilation

- ❖ Note the rapid rise to peak flow almost instantly, followed by deceleration (decay).
- ❖ When the flow decelerates to 25% of the peak flow, the flow changes to expiration and expiration is started.

- Hence, pressure support ventilation (PS) is **a flow-cycled mode,** because the inspiration ends on the basis of a *flow criterion* (when 25% if the peak flow is reached), not *pressure*, *time* or *volume* (Refer to the cycling in the beginning of this Chapter, and Chapter 7 Mechanical Ventilation).
- This feature *allows the patient to retain control over the respiratory rate, inspiratory time, and volume.*
- PS mode may be used in conjunction with SIMV mode. In that case, only the spontaneous breaths will be pressure supported and the mandatory breaths are not.
- The great advantage of combining PS with SIMV is that, if the patient becomes apneic, he is assured of the preselected back number of mandatory breaths.
- It can be used either with a high level pressure support or low level pressure support.
- In high level (PS_{MAX}), the PS is increased till the patient gets a tidal volume of 10 to 12 ml/Kg. With this support, the patient doses not require any further support with volume cycled breaths, provided he has enough and consistent respiratory drive.
- In low-level PS, the amount of support is adjusted to achieve a tidal volume (V_T) of 5 to 8 ml/Kg. This low level PS can be used alone or it can be combines with SIMV.
- When PEEP is combined with PS, (it is called PEEP and not CPAP, as it is combined with this mode of ventilation), the peak inspiratory pressure (PIP) is equal to the sum of both. If the PEEP applied is 5 cm H_2O and the PS applied is 8 cm, then the PIP is 13 cm H_2O.
- To institute pressure support ventilation, the patient should have ***a strong reliable respiratory drive*** and the ***ability to consistently trigger the ventilation*** because all the breaths are patient triggered.
- Ideally initial pressure support level is chosen in such a way that it unloads the respiratory muscles completely.

- When the condition improves and the disease resolves, the pressure support level can be gradually decreased to allow the patient's respiratory muscles to assume greater levels of work of breathing. Mechanical ventilation is discontinued when the pressure support level is brought down to about 6 cm H_2O.

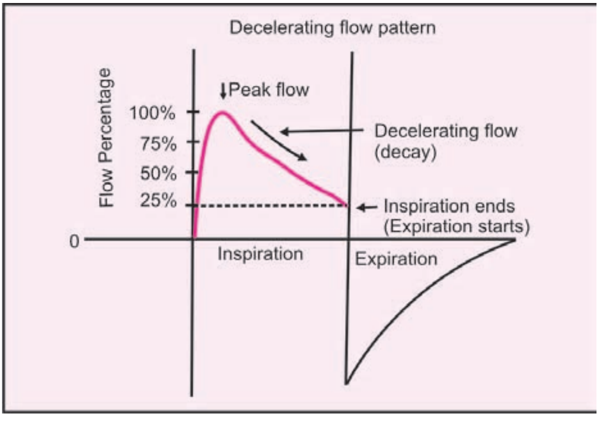

Fig. 9.10D: The full flow pattern of pressure support ventilation

❖ Note the decelerating flow with initial peak flow which decelerates towards the end of inspiration.

❖ By the time the flow decays to 25% of the peak flow, the ventilator ends the inspiration and starts the expiration.

❖ The ventilator opens the expiratory port and expiration and expiratory flow is seen in the opposite direction.

Indications

- This mode is used for weaning the patient from ventilator, because the amount of work imposed to the respiratory muscles can be tightly controlled by adjusting the level of PS.
- By augmenting the inspiratory flow, PS reduces the WOB associated with artificial airways as endotracheal tube and ventilator circuitry.

Advantages

- It will overcome the additional work of breathing imposed by the resistance of artificial airways and ventilator circuit, for moving the air during inspiration.
- The reduced work of breathing will decrease the oxygen required for the work, and this in turn will make the patient tolerate weaning.
- In this mode patient initiates his inspiration, has control of the timing of inspiration and expiration, and the rate. Because of this, it improves patient comfort and patient-ventilator synchrony.
- The operator could augment inadequate tidal volume (V_T) to any desired degree and set the PIP.
- The support provided to the patient is 'titratable' than with any volume cycled ventilatory modes (As seen earlier, this is a flow-cycled mode).
- Every spontaneous breath is assisted and the amount of assistance can be reduced in increments as small as 2 cm H_2O, thereby gradually titrating the amount the support withdrawn from the patient.
- It can be compared to SIMV; when the number of mandatory breaths is reduced, the work so far done by the machine is fully transferred to the patient's respiratory muscles during weaning.
- The mean airway pressure is lower, because the PIP is generally lower than with volume cycled ventilation.

Disadvantages

- As discussed earlier, the tidal volume is variable and so the alveolar ventilation is not guaranteed.
- If there is an increase in resistance or decrease in compliance due to patient factors or ventilator circuit factors, the tidal volume will decrease. Hence PS must be

cautiously used in patient showing significant increase in impedance due to bronchospasm or secretions.

- The ventilator may fail to cycle to expiration, if there is leak in any part of the system because the flow rate that terminates the inspiratory pressure support will not be reached. It is a vital aspect to be remembered.

Points to Consider and Monitor

- Monitor the EV_T carefully. When full ventilatory support is given, a tidal volume of 10 to 15 ml/kg is used and for partial support, 5 to 8 ml/kg is used. If there is a fall the cause must be identified and corrected. Otherwise rapid development of atelectasis may be caused.
- Tidal volume can be increased by increasing the amount of PS.
- If the patient is in weaning trial, and the increase in respiratory rate (RR) and decrease in the EV_T is a sign of fatigue. Therefore, the patient must be given more support by increasing the PS level.

Volume Assured Pressure Support

- This is a modification of PSV mode.
- The patient breaths spontaneously at pressure support level set by the clinician.
- If the tidal volume fails to reach the preset minimum value, chosen by the clinician, an additional constant flow guarantees the minimum tidal volume.
- During the inspiratory phase, two sources of flow will be working in parallel; one system with a fixed square wave which can deliver a constant, adjustable flow what ever the airway pressure is and a second demand flow with no flow limitation designed to maintain the airway pressure at the preset pressure support level.

- At the beginning of the flow, both the flows are activated and they remain so until the preset tidal volume is delivered.
- After this the constant flow stops and if the patient's spontaneous effort continues the demand flow is available to complete the inspiration.
- Thus in VAPS mode, the patient is allowed to generate a tidal volume higher than the set minimum tidal volume if he is capable of it and if he cannot generate the minimum tidal volume, the constant flow ensures the delivery of preset tidal volume.
- This mode may be useful to maintain a minimum minute volume in patients with variable levels of respiratory drive and varying lung mechanics.

Pressure Control Ventilation (PCV)

Ventilator determines inspiratory time – no patient participation

- Triggered by time
- Limited by pressure
- Affects inspiration only
- In this mode, there is *a preset respiratory rate* (RR) and every breath is augmented by *a preset amount of inspiratory pressure*.
- *Functionally, pressure controlled ventilation (PCV) is similar to controlled mandatory ventilation (CMV) in that, the clinician sets the respiratory frequency (RR), inspiratory time and PEEP as in CMV.*
- The difference is, here in PCV the clinician sets the airway pressure to be maintained during the inspiratory phase.
- The rapidity of pressure rise at the beginning of inspiration is determined by the pressure control slope set by the clinician (Fig. 9.11).

- Each breath is time triggered and the patient is unable to trigger the ventilator to take additional breaths above the preset rate. This is achieved by setting the sensitivity in such a way that it will not respond to the patient. Therefore, *each breath is a machine cycled mandatory breath* (Fig. 9.11).

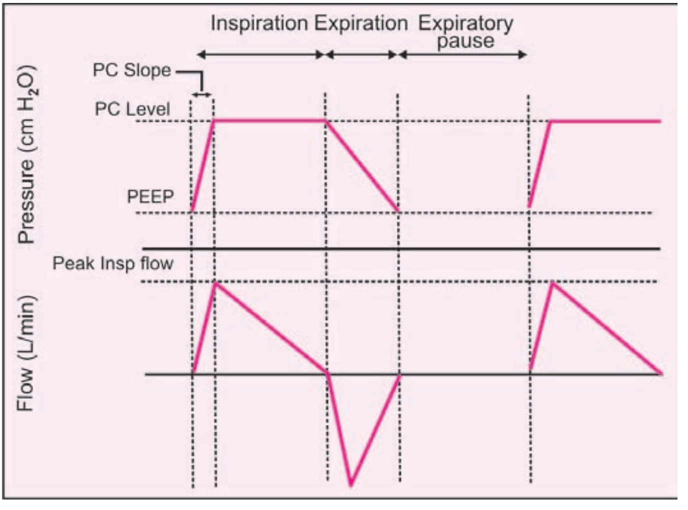

Fig. 9.11: The pressure control ventilation (PCV)

❖ Note that all breaths are mechanical breaths; patient does not initiate any breath.

❖ The pressure control slope, the rate (RR), and the inspiratory pressure are set by the physician.

- However, PC may also be applied with a sensitivity that will respond to the patient, allowing additional breaths to be triggered. These additional patient triggered breaths called *'machine cycled assisted breaths'* are also augmented by the preset amount of pressure.
- Here the tidal volume (V_T) is not preset. *The tidal volume in each breath is variable* and is determined by the set

inspiratory pressure, the RR, the inspiratory time, pulmonary compliance, and airway and circuit resistance.

- The onset of inspiration is primarily by the timing mechanism.
- Once initiated, the inspiratory flow of gases is augmented by the preselected amount of pressure that is the PC level.
- The flow is delivered by *a decelerating flow wave pattern*. With the decelerating flow pattern, the flow rate naturally decays as the patient's lungs fill with air on inspiration. Unlike pressure support (PS), where the cycling is done by the decay of flow rate (flow cycling), here in PC it is time cycled to end inspiration and begin expiration.

Indications

- It may be used as a method of providing full ventilatory support in patients with noncompliant lungs who exhibit high airway pressure and poor oxygenation while supported by volume cycles ventilation.
- Pressure control can bring the airway pressure under the control of the clinician.

Advantages

- It is believed that the pathology in ARDS results in high PIP during ventilation with volume cycled ventilation. This higher PIP is one of the causes for barotrauma. PC ventilation is postulated to reduce the barotrauma due to high ventilating pressures and uneven gas distribution.
- Reduced airway pressures are promoted by limiting the inspiratory pressures to that level which achieves the

desired tidal volume (V_T). This pressure is definitely much lower than that is generated in volume cycled ventilator.

- The decelerating flow wave pattern of PC ventilation changes the nature of flow in the lungs and promotes *laminar flow* of gases which is more ideal for even distribution of gases.
- The laminar flow wave, because of the parabolic curve in the flow head (discussed in mechanics of breathing) wedges their way into the airways and alveoli, creating less airway trauma and more uniform gas distribution.
- Decelerating flow wave has been associated with a significant reduction in total resistance, improved pulmonary compliance, a decrease in dead space ventilation, and an increase in oxygenation.

Disadvantages

- Requires frequent adjustments to maintain adequate minute ventilation (V_E).
- Pt with noncompliant lungs may require alterations in inspiratory times to achieve adequate $V_{T.}$
- Monitoring of the applied pressure must be done. The main disadvantage of a pressure mode like pressure control mode (PC) is the reduction in preload to the right heart and thereby a reduction in cardiac out put.
- Appropriate measures must be taken to improve the preload and any support needed with inotropic agents. Otherwise the patient will lose the benefit of improved oxygenation and PaO_2 as the overall tissue oxygen delivery is compromised.

Points to Consider and Monitor

- Rate of respiration must be monitored, as any increase will cause hyperventilation and respiratory alkalosis.

- Exhaled tidal volume (EV_T) and minute volume (V_E) must be closely monitored. Any increase in resistance or any reduction in compliance will adversely affect the tidal volume. Similarly, with improvement in lung characteristic, if the pressure is not reduced correspondingly, overdistension and hyperventilation will be caused.
- The PIP must be equal to the PC level and any applied PEEP.
- Hemodynamic status must be monitored with an anticipation of compromise due to increase in mean airway pressure (MAP).

Inverse Ratio Ventilation

The reason for discussing this method of ventilation immediately after discussing Pressure control ventilation (PC) is, because it is commonly employed along with PC.

As seen earlier, in pressure control (PC) *there is a preset RR and every breath is augmented by a preset amount of inspiratory pressure.* This is an additional strategy used with mode of ventilation if further improvement of oxygenation is needed.

A set rate, a set support of inspiratory pressure, and inversion of I : E ratio

- This refers to mechanical ventilation in which the I:E ratio is greater than **1.**
- By increasing this ratio to more than one, the inspiratory time is prolonged and consequently expiration time is reduced.
- The I : E ratio can be inverted as 1 : 1, 2 :1, 3 :1, 4 :1, in such a way that the inspiratory time equals or exceeds the expiratory time.
- It is *to improve gas distribution* in the lung and increase mean airway pressure for *improvement of oxygenation*.

- In addition to changing the inspiratory time and expiratory time, the RR must be set sufficiently high, so that the patient does not exhale completely before the initiation of next breath. This will result in air trapping in the lung (auto-PEEP), which keeps the critical closing volume above the point of alveolar collapse (Fig. 9.13).
- As the ventilatory pattern is modified, *sedation and paralysis by using a muscle relaxant are usually necessary to maintain patient-ventilator synchrony.*
- So, the basic requirement for a inverse ratio ventilation are; use of a critical rate of respiration to promote formation of auto-PEEP and sufficient inspiratory pressure to overcome the opening pressure of the lung. Both are present with pressure control ventilation (PC).

Indications

- In surfactant deficient diseases like ARDS where the abnormality is diffuse and nonuniform throughout the lung.
- This is one of the methods of providing full support of ventilation for patients with noncompliant lung who exhibit high airway pressure and poor oxygenation when volume cycled ventilators are used.

The effects of inversing the ratio on ventilation has been discussed in the previous chapter. However it is briefed here.

Effects of Increased Inspiratory Time

- Increase in mean airway pressure (MAP) without increasing peak pressure despite a constant tidal volume and PEEP level is caused by reduction in the inspiratory flow for the given tidal volume, acting to open collapsed alveoli.

- The lung units with 'longer time constant' ("Slow" lung compartments) will not approach full inflation during an inspiratory time of standard duration. Moreover, these poorly inflated alveoli require a more sustained pressure for longer time to open up. This reversed inspiration time which is longer, causes sustained alveolar inflation, and also decreases the dead space in the diseased lung, by enhancing the efficacy of collateral ventilation.
- In ARDS, alveoli with relatively more disease have longer time constants and require more time to fill. In normal ventilation, there is not enough time for these alveoli to fill and so remain in a collapsed state, resulting in persistent intrapulmonary shunt and hypoxemia.
- The IRV will give adequate time for filling the diseased alveoli and improve overall distribution of gas in the lung.
- The inspiratory time encroaches into the expiratory time, so the alveoli have less time to empty and air trapping occurs, which is known as "dynamic hyper-inflation" resulting in auto PEEP. Auto-PEEP splints the unstable alveoli at end of expiration, prevents them collapsing.

Effects of Decreased Expiratory Time

- This builds up an auto-PEEP, because the tidal volume cannot be exhaled fully before the next inspiration is started (Fig. 9.13).
- This internal PEEP prevents the slow compartments from collapsing, resulting in an increase in FRC. This improves oxygenation, by reducing shunt (unventilated alveoli).

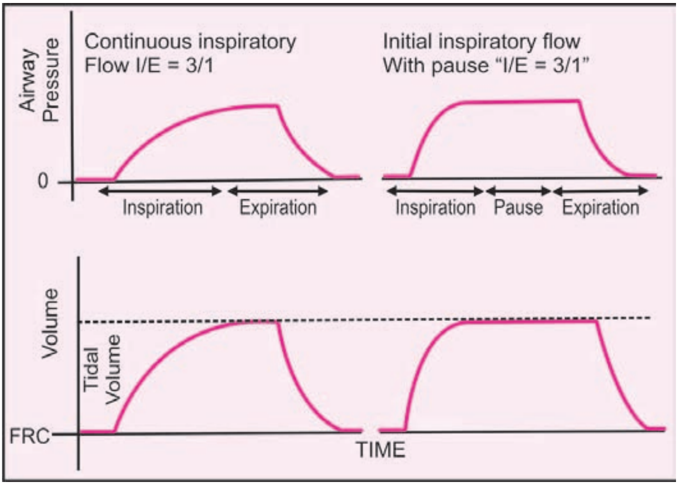

Fig. 9.12: Inverse ratio ventilation

- ❖ Two methods of achieving the inverse ratio.
- ❖ Showing the pressure and the volume changes.
- ❖ Note that in the second method with initial flow with pause it is convenient to reduce the pause gradually while withdrawing IRV.

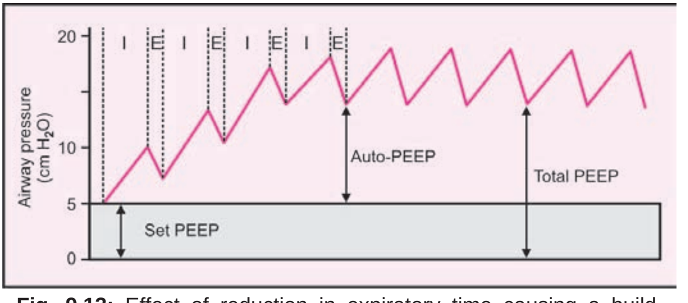

Fig. 9.13: Effect of reduction in expiratory time causing a build up of internal PEEP

- ❖ Note the inversion of I : E ratio.
- ❖ The shades height above the baseline shows the set PEEP.
- ❖ The height from set PEEP to the ends of expiration after applying IRV is auto-PEEP.
- ❖ Insufficient expiratory time permits air trapping in the lung and that cerates built up pressure, known as "auto-PEEP".

- However, external PEEP is necessary in addition to the internal PEEP caused by IRV, as the external PEEP is very important for the stability of the faster compartments of lung that empty completely with IRV.
- *An I : E ratio of 2 : 1 is rarely exceeded* as, It may not be required beyond this ratio for improving oxygenation and for fear of potential adverse hemodynamic effects.
- Once the oxygenation starts improving, the inspiratory time may be gradually reduced by decreasing the length of *the inspiratory pause* (Fig. 9.12).

Airway Pressure Release Ventilation (APRV)

- This mode was designed to allow the patient to breathe spontaneously with CPAP while mechanically augmenting the alveolar ventilation *by brief interruption of CPAP.*
- Essentially, this consists of a high flow CPAP system with release valve in the expiratory limb. Opening of the valve for a brief period (1 to 2 secs) causes a drop in the airway pressure usually to a new lower level of CPAP. The magnitude of drop of pressure is called as the 'release pressure' (Fig. 9.14).

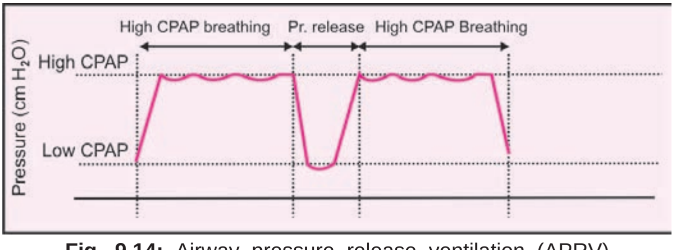

Fig. 9.14: Airway pressure release ventilation (APRV)

❖ Note the high CPAP level in which the patient is breathing spontaneously.

❖ The high CPAP is momentarily dropped to the low level CPAP and again raised to the original high CPAP.

- The patient is breathing spontaneously on one level of CPAP. After a set period of time the CPAP level is released and allowed to drop to a constant level just above the baseline maintaining resting lung volume above FRC.
- The inspiratory flow valve is open throughout the ventilatory cycle so that the patient is able to breathe spontaneously in the similar manner as in CPAP.
- However, at the preset intervals, the positive airway pressure applied by the ventilator at the airway opening is released and the lungs are allowed to deflate to a level just above the FRC, determined by a preset end expiratory pressure. It can be atmospheric pressure also (0 cm H_2O).
- But the low CPAP must be decided initially before instituting APRV, because this should be equal to the CPAP which maintains an optimal SaO_2 (at least 95%) at an F_IO_2 no greater than 0.5.
- This short pressure release expirations – the release times (about 0.5 to 1.5 seconds) is sufficient to complete exhalation without air trapping. By this, CO_2 elimination is improved. 0.5 sec is enough for most clinical situations.
- The return to the original CPAP level is provided by the machine.

The Control Settings

- Upper CPAP
- Lower CPAP
- Pressure release time
- Frequency of the release.

Advantages

- Augmentation of alveolar ventilation with low peak airway pressure without overdistension of lung is the major advantage.
- Low intrathoracic pressure with better matching of ventilation and perfusion.

Biphasic Positive Airway Pressure (BIPAP)

- It is also known as Bi-level positive airway pressure ventilation
- This is another variant of APRV mode. In this the patient is allowed to breathe spontaneously at two levels of CPAP.
- It is described as a CPAP system with a time cycled change of the applied CPAP level (Fig. 9.15).
- The duration of high and low level breathing and the two levels of CPAP are infinitely adjustable.
- In CPAP high pressure level requires increased respiratory effort for patient during expiration which can result in muscle fatigue and reduction in compliance. Hence, the patient on CPAP ventilation, the CPAP was reduced during expiration to facilitate easy exhalation by the patient.
- Hence, for the patient on BIPAP ventilation, the CPAP was reduced during expiration to facilitate easy exhalation by the patient. Inspiratory CPAP (high) level is called IPAP and expiratory (low) CPAP is called EPAP resulting in Bi-level ventilation (Fig. 9.15).
- Flow sensors in the machine sense both the inspiratory and expiratory signals from the patient and alternatively switch to EPAP to IPAP and *vice versa*. Presently this is known as Bi-level patient mode. This prevents patient-machine asynchrony.

- Later on twin trigger mechanism is introduced in the above unit which can trigger the next inspiration, if the patient fails to take a breath himself within the period of time set by the operator. This is known as Bi-level twin trigger.
- As in the CPAP the patient can breath spontaneously both during EPAP and IPAP.
- The work of breathing is decreased proportionate to the magnitude and the frequency of the pressure release.
- By choosing a pressure release level that provide s an optimal tidal volume and setting the pressure release at around 12 to 15 breaths, it is possible to ventilate even an apneic patient.
- As the spontaneous respiratory function of the patient improves, the frequency and magnitude of pressure release can be gradually decreased until the patient fully breaths spontaneously.

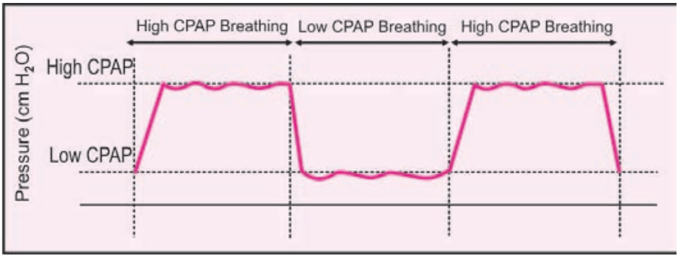

Fig. 9.15: Biphasic positive airway pressure ventilation (BIPAP)

Advantages

- Improves alveolar ventilation without sacrificing oxygenation.
- Minimises the intrathoracic pressure and reduces the need for sedation while maintaining CO_2 elimination.
- Resumption of spontaneous breathing by the patient does not require changing of mode of ventilation.

Proportional Assist Ventilation (PAV)

- Microprocessor is developed to detect the patient's activity and producing appropriate ventilator response.
- This improves patient ventilator synchrony.
- PAV is a form of synchronised partial ventilatory support in which the ventilator generates pressure in proportion to the patient's effort.
- The more the patient pulls, the more pressure the machine generates.
- This is under trial. The responsibility of determining the level and pattern of the breathing is shifted to the care giver.

Mandatory Minute Volume (MMV)

- This is another combination of spontaneous breathing with mechanical ventilation.
- It is a Servo controlled mode similar to SIMV.
- Instead of predetermined rate of ventilation (SIMV), predetermined minute volume is the criterion.
- The ventilator measures the patient's actual minute volume and compares to the minimum minute volume set by the clinician.
- The difference in the two is delivered as mandatory breath by the ventilator by a tidal volume and flow that was set by the clinician.
- A "mandatory" minute volume (MMV) is preselected and a fresh gas flow (FGF) equal to this is fed into a constant pressure reservoir from which the patient breathes.
- If the patient breathes more than the preset FGF, a valve system permits additional FGF.
- If the patient's spontaneous minute volume falls short of the preset MMV, the excess gas is collected in

bellows and when the gas collected in the bellows reaches a preset value, a mechanism triggers the ventilator to deliver the contents of the bellows into the patient's lungs.

- If the patient stops breathing, the total MMV will be delivered to the patient in preset number of mechanical breaths with preset tidal volumes.
- If the patient's spontaneous ventilation equals to the MMV, then no mechanical breaths will be delivered.
- Unlike in SIMV, the rate of mechanical breaths varies from minute to minute.
- However, this system is theoretically excellent and does not work as well as it sounds, because the patient may cheat it by adopting an undesirable pattern of rapid, shallow breathing which can give a minute volume equal or greater than the preselected MMV.
- Useful in patients weaned from ventilator, but have fluctuations in their ventilatory drive.

Permissive Hypercapnea

- In ARDS patients, with conventional mechanical ventilation barotrauma is a potential problem. Permissive hypercapnea is one among the various methods tried to decrease the chances for barotrauma.
- It is a ventilatory strategy of reducing the tidal volume and accepting a certain degree of hypercapnea and associated respiratory acidosis with out attempting to correct it. This is termed as "permissive hypercapnea".
- Hypercapnea will cause significant cardiovascular effects such as increase in stroke volume, cardiac output and pulmonary artery pressure.
- There is a possibility of a decrease in PaO_2 due to two reasons, a decrease in alveolar oxygen tension and

alveolar derecruitment (progressive collapse). However, the increased cardiac output mediated through sympathetic stimulation compensates this to some extend.

- Similarly, the PEEP if applied causes a permanent recruitment of alveoli with low tidal volume ventilation.
- Permissive hypercapnea is contraindicated in patients with ischemic heart diseases as it may cause coronary steal. In patients with intracranial pathology, it may increase the cerebral blood flow and intracranial tension.

Automatic Tube Compensation

- This is a method used to assess the readiness of the patient breathing spontaneously whether he can tolerate extubation.
- During spontaneous breathing, as the inspiratory flow increases, the airway resistance increases and as a result the pressure drop across the endotracheal tube also increases.
- During this, if a fixed level of pressure support is applied, it may under compensate during inspiration and overcompensate during expiration.
- Automatic tube compensation (ATC) will precisely compensate for the resistance of the tracheal tube without under or over compensation throughout the cycle.
- The pressure gradient across the ends of the given endotracheal tube of a given internal diameter can be calculated for different flow rates.
- If this detail of pressure gradients for various flow rates and the diameter of the endotracheal tube are provided, the ventilator can constantly alter the level of pressure

support during a single breath as the patient's flow rate varies from moment to moment.

- In fact the ventilator can provide exact amount of pressure support needed to overcome the resistance at each instant of inspiratory phase. This will give an opportunity to assess the ability of the ventilated patient to breathe spontaneously without ventilatory support. Hence, it is known as *'electronic extubation'*.
- To set the ATC, the clinician enters the size of the tube and chooses the percentage of compensation in arrange from 1 to 99% depending upon the indented load which can be left on the patient's muscles. Under compensation may be used for training the patient's respiratory muscles.
- Some ventilators have ATC for expiratory phase also.
- Because this strategy is not available in all ventilators and the measurements of respiratory mechanics as a routine during ventilatory support is difficult, this is not routinely used.

NONCONVENTIONAL MODES OF VENTILATION

Conventional ventilation therapy for patients with respiratory failure was always used intermittent positive pressure ventilation (IPPV) and positive end expiratory pressure (PEEP). Some patients, in spite of this treatment do not improve, and some of them deteriorate even after adding high level of PEEP to improve oxygenation and when large tidal volume was used peak inspiratory pressure (PIP) rises.

This necessitated the search for other modes of ventilation for improving the patient's condition. The two main categories are High frequency ventilation and Independent lung ventilation. Here only the High frequency ventilation is discussed.

High Frequency Ventilation Techniques

In conventional ventilation near physiological respiratory rates (RR) and tidal volumes greater than the anatomical dead space were used.

It has been proved by extensive studies that adequate arterial oxygenation and alveolar ventilation could be achieved with the use of substantially smaller tidal volume (1 to 5 ml/kg) and higher respiratory rates than conventional methods (60 to 3600 breaths/minute)

This group of ventilatory techniques using higher rates and low tidal volume delivery do not try to reproduce the mechanics of normal spontaneous breathing are collectively called as High frequency ventilation.

The frequency ranges from 60 to 3600 cycles/minute.

The three types known are:

HF	Positive Pressure Ventilation	(HFPPV)	Cycles	60-100/min.
HF	Jet Ventilation	(HFJV)	Cycles	100-300/min.
HF	Oscillatory Ventilation	(HFOV)	Cycles	300-3600/min

Among these the High frequency Jet ventilation is the only method commonly employed.

High Frequency Jet Ventilation (Fig. 9.16)

- This is different from other high frequency ventilations in the mechanism by which the gas is delivered.
- It is the delivery of a small tidal volume of 3.5 to 4.5 ml /kg at a high breath frequency of 60 to 150 breaths/ min through a small endotracheal catheter or cannula placed in the airway.
- The volume is delivered under considerable driving pressure, about 20 to 50 ponds/square inch, through a cannula of 1 to 2 mm diameter.
- This cannula is commonly refereed to as “the injector” and is usually a 16 or 14 gauge needle.

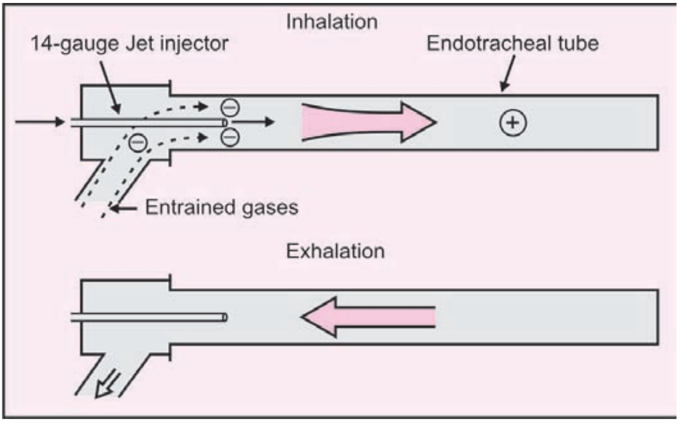

Fig. 9.16: The high frequency jet ventilator injector (HFJV)

- ❖ Through a 14-gauge jet injector (a narrow tube) fresh gas is allowed to flow at an accelerated rate as a pulsing jet during inspiratory cycle.
- ❖ According to "Venturi" principle, when the velocity of gas in a tube increases, there is fall in the pressure it exerts on the wall of the tube proportionately.
- ❖ A negative pressure is thus created at the outer aspect of the tip of the 'jet' injector.
- ❖ This negative pressure allows gases from neighboring areas to enter (by suction effect) through the entrainment port provided at the side.
- ❖ Humidified, low flow (20 L/min) gases are entrained in this area of negative pressure, continuously replacing the gases that have moved forward.
- ❖ This rapidly causes inflation of the lungs by moving the gasses into the lungs.

- The 'jet' works on the Venturi principle and the bulk of air enters into the trachea by way of entrainment of humidified additional fresh gas.
- The volume delivered is a combination of insufflated gas through the jet needle and the gas which is entrained by venturi.

- The volume delivered is targeted as two or three times the minute volume (V_E) delivered by conventional ventilation.
- The delivery of a very small tidal volume (V_T) at rapid rate results in a constant flow, low pressure ventilation.
- The theoretical advantages of HFJV are its ability to lower peak airway pressures and to improve the efficiency of the ventilation. This results in less pulmonary barotrauma.
- Ventilation efficiency is possibly achieved by the big minute volume about two or three times the conventional ventilation which enhances the CO_2 elimination.
- It is reported to be better tolerated by the patients and less sedation is required. More over there is smaller changes in intrathoracic pressures.
- However, the machine makes a loud sound when it works which disturbs the patient and the environment.

Gas Exchange in HFJV

- The physiological mechanism by which the alveolar ventilation and oxygenation are enhanced in the HFJV is not well understood. However two possible mechanisms are proposed.
- *Convection* is the process in which the flowing molecules carry the other molecules along with them. In jet ventilation convection plays a major part in moving the gases.
- When the gases exit from the tip of the injector cannula, the pulsed gases acquire characteristics of a jet stream. The kinetic energy of the jet stream is transferred to the immobile gases present in the airway and makes them move by convection towards the distal airway.

- Another factor is *enhanced diffusion*. The gas mixing and diffusion are augmented by the high flow of gases which cause turbulence and total movement of molecules.
- Oxygenation is well maintained in HFJV because of an auto PEEP created. High rates produce inadequate emptying and trapping of gases in the alveoli. This causes an increase in end expiratory pressure (PEEP).
- This auto PEEP causes an increase in FRC, alveolar stability and improved oxygenation.
- It can be recalled that, the FRC and the mean airway pressure are the main determinants of adequacy of oxygenation what ever mode of ventilation is used.
- Mean airway pressure is not significantly reduced in this mode, so the hemodynamic consequences of a higher mean airway pressure are similar to conventional ventilation.

Indications

- The main indication is while doing bronchoscopy.
- Ventilation of postoperative patient, after pneumonectomy, to avoid stress on the sutured bronchus.
- Combined head injury and respiratory failure, because reduced intrathoracic pressure will result in reduction of intracranial pressures.

New Generation

There are other high frequency modes added recently. They are:

- Ultra-high Frequency Jet Ventilation (UHFJV).
  Near resonance frequency.
- HF Negative Pressure Cuirass Ventilator.

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CHAPTER 10

Acid-base Regulation

- *Acid-base regulation*
- *Respiratory acidosis*
- *Respiratory alkalosis*
- *Metabolic acidosis*
- *Metabolic alkalosis*
- *Interpretation of arterial blood gas values*
- *Technique of obtaining arterial blood samples*

The basic functions of respiratory system are,

- Maintaining adequate oxygen saturation of blood (PaO_2) and supplying oxygen to the tissues.
- Elimination of CO_2 from the lungs so as to maintain $PaCO_2$ in the normal rage.
- Maintaining the acid base status of blood in the normal range.

ACID-BASE REGULATION

The Respiratory System The Renal System	Work constantly to keep the Acid-base status of the body normal to provide the optimal internal environment – for proper function of metabolic process.

Acid-base Status

This is a reflection of H^+ ion concentration in the body – which is represented by the **pH.**

- When **H^+ ion** concentration increases the **pH** falls.
- When **H^+ ion** concentration increases the **pH** rises.

The normal arterial pH is **7.35 to 7.45** and is maintained by a balance of acid to base in the body.

- **Acid** is a substance which dissociates in solution to **liberate H^+ ion.**
- **Base** is a substance which can bind or **accept H^+ ions**.
- Normally the body has 20 acid ions for every base ion.

This relationship can be calculated with the Henderson-Hasselbach equation.

Henderson-Hasselbach Equation

$$\textbf{pH} = \text{pK} + \text{Log}\ \frac{[HCO_3^-]}{PaCO_2} \text{ or } \frac{\text{Kidneys}}{\text{Lungs}} \text{ or } \frac{20}{1}$$

Where **pK** is a constant **6.1**

This equation defines the relationship between **pH, $PaCO_2$, and Bicarbonate (HCO_3^-)**

- Bicarbonate is regulated mainly by Kidneys.
- CO_2 is regulated by lungs.

The ratio of bases to acids must remain at 20 : 1 to maintain the normal pH.

- When this ratio becomes imbalanced, resulting in an increase in H^+ ions, the pH decreases < 7.35, then the patient is in **Acidemia.** This process is called **Acidosis.** For example, Diabetic Ketoacidosis.
- When the ratio of acids to bases in the body is imbalanced, leading to an excess of bases, the pH increases > 7.45. Then the patient is in **Alkalemia.** This status is called as **Alkalosis.**

H^+ ions (Hydrogen ions) are added to the body fluids as a byproduct of metabolic processes. These acids have to be either eliminated or neutralized by the body, so that the patient does not develop acidemia.

The H^+ ions or acids produced are either,

- Fixed Acids: Hydrochloric acid or carbonic acid, are to be excreted and eliminated by kidneys.
- Volatile Acids: Carbon dioxide is a volatile acid-eliminated through the lungs by means of adequate alveolar ventilation.

So the lungs and kidneys are the primary organs for the maintenance of Acid-base Balance.

To some extent, both these volatile and fixed acids are neutralized in the body through combination with a base.

Similarly, strong bases may be neutralized through combination with weak acids.

These two processes are called ***Chemical Buffering.***

Chemical Buffers are therefore, substances that minimize changes in pH when either acids or bases are added.

Buffer systems in the body are present in various locations

- Proteins and phosphates are buffers in the cells.
- Hemoglobin is a buffer in the RBCs.
- Bicarbonates and proteins are buffers in the extra cellular fluid.

All these are called as ***"Total Buffer Base".***

The most important buffer system which accounts for more than half of the total buffering is ***"Bicarbonate Buffer System".***

Lungs **Kidneys**

$$\begin{array}{ccccc} \textbf{Lungs} & & & & \textbf{Kidneys} \\ \uparrow & CA & & & \uparrow \\ CO_2 + H_2O & === & H_2CO_3 & === & H^+ + HCO_3 \end{array}$$

Carbonic acid (H_2CO_3) is formed by the combination of (CO_2) to water (H_2O) in the presence of the enzyme carbonic anhydrase (CA). Carbonic acid quickly dissociates into H^+ hydrogen and Bicarbonate (HCO_3).

The bicarbonate buffering system then operates by using the lungs to regulate CO_2 and the kidneys to regulate HCO_3^-.

The respiratory system controls the carbon dioxide (CO_2) tension of the blood by regulating alveolar ventilation. This process may very quickly correct the acid-base disturbances.

- ***Alveolar Ventilation*** **(V_A)** is the volume of gas within the respiratory bronchioles and alveolar duct, i.e. Respiratory zone of lung.

  V_A dose not reflect the volume of gas moved in and out of upper airway (mouth).
- ***Minute Ventilation*** **(V_E)** is the volume of gases into and out of upper airway (mouth) per minute.

  This consists of Alveolar Ventilation (V_A) and Dead space ventilation (V_D)

  Therefore alveolar ventilation $V_A = V_E - V_D$. Alveolar ventilation strives to maintain the $PaCO_2$ at 35–45 mm Hg (Eucapnia).

  Hypoventilation or decreased alveolar ventilation, results in excessive acids. When the $PaCO_2$ is > 45 mm Hg, ***Hypercapnea*** is present.
- If the pH is < 7.35 and the $PaCO_2$ is > 45 mm Hg, the patient is said to be in "***Respiratory Acidosis*".** Hyperventilation, or increased alveolar ventilation, causes the acids to be blown off.
- When the $PaCO_2$ is < 35 mm Hg and the pH is > 7.45, Hypocapnea is present and this condition is called as ***Respiratory Alkalosis.***
- It is important to note that ***Hyperventilation*** is not synonymous with a ***rapid respiratory rate.*** For example, the patient may be breathing 40 times per minute and still have a $PaCO_2$ of 50 mm Hg.
- The kidneys defend the blood pH by controlling bicarbonate concentration. This is accomplished:
  - By excretion of hydrogen ions in the urine when the blood is too acidic.
  - By excretion of bicarbonate in the urine, when the blood is too alkaline.

It may take ***many hours to days*** for the kidneys to affect the pH. So,

- Rapid compensation is by Respiratory System.
- Slow compensation is by Renal System.

- If there is abnormal rise in HCO_3^- concentration in the serum or a significant loss of hydrogen ions, accompanied by a pH of > 7.45, the patient is said to be in ***Metabolic Alkalosis.***
- If there is a loss of bicarbonate or rise in hydrogen ions or both accompanied by a pH of < 7.35, the patient is said to be in ***Metabolic Acidosis.***

Compensation: refers to a return of the blood pH back to normal by the lungs or kidneys.

The system, respiratory or renal opposing the primary disorder will attempt the compensation. For example, in respiratory alkalosis, the body will, first attempt to compensate by decreasing Alveolar Ventilation. ***However, hypoventilation cannot occur to a significant degree, because hypoxemia will eventually stimulate the drive to breath.***

The renal system will then attempt to compensate for the respiratory acid-base imbalance by excreting HCO_3^-.

If the compensation is ***complete***, the pH will return to normal. If the compensation is partial, then the pH will be working its way towards normal. The body does not over compensate.

THE COMMON CAUSES, SYMPTOMS AND TREATMENT OF ACID-BASE DISTURBANCES

Respiratory Acidosis

Causes

- Hypoventilation due to any cause.

- Acute process aggravating chronic lung diseases, severe obesity, respiratory centre depression, e.g. Stroke, Head injury, Drug overdose, etc.
- Respiratory or neuromuscular disease and airway obstruction.

Symptoms

- Acute CO_2 retention, confusion, lethargy, stupor, coma.

Treatment

- Treating the hypoventilation by treating the underlying cause.
- Making the patient cough and do deep breathing.
- Incentive spirometry, bronchodilators and if needed, intubation and mechanical ventilation.

Respiratory Alkalosis

Causes

- Hyperventilation due to any cause.
- Hypoxemia, anxiety reaction, CNS irritation, e.g. Central hyperventilation.
- Metabolic acidosis.
- Excessive artificial ventilation.

Symptoms

- Complaints of shortness of breath, anxiety, muscle cramps, tetany, perioral tingling, seizures.

Treatment

- Decrease alveolar ventilation, sedation, improve oxygenation, rebreather bag, change ventilator settings (Decrease Rate or Tidal volume).

Metabolic Acidosis

Causes

- *Excessive acids* as in, diabetic ketoacidosis, renal failure, lactic acidosis, starvation, salicylate overdose, ethylene glycol.
- *Bicarbonate loss* as in, diarrhea, pancreatic, biliary, or small bowel fluid loss, renal disease.

Symptoms

- Kussmaul respirations (deep, rapid), disorientation, restlessness, coma.

Treatment

- Treating the underlying abnormality.
- In bicarbonate loss, replace HCO_3^-.

Metabolic Alkalosis

Causes

- *Loss of acids* as in nasogastric suction (chloride also is lost).
- *Excessive base* as in overdose of antacids, milk of magnesia, or $NaHCO_3^-$, citrate in blood transfusions, lactate in hyperalimentation.
- *Diuretic therapy:* resulting in K^+, Na^-, and Cl^- losses. H^+ ion moves into cells, HCO_3^- concentration increases.

Symptoms

- Apathy, mental confusion, shallow breathing, tetany, spastic muscles.

Treatment

- Control of emesis or GI losses.
- If unable to control, replace chloride with Ringer's Lactate solution or Sodium chloride.
- Chloride replacement allows HCO_3^- to exit the cells to be excreted.
- Reduce the use of alkaline antacids, monitor diuretic use, and administer acetazolamide to increase renal HCO_3^- excretion, correct potassium depletion.

INTERPRETATION OF ARTERIAL BLOOD GASES

- Using the information about respiratory chemistry presented, for a given arterial blood gas (ABG) measurement, it is possible to determine whether the primary disturbance in the acid base status is due to ***Respiratory*** or ***Metabolic*** reasons.
- An ABG measurement provides more information than the acid base balance.
- ABG, apart from providing information ***about acid-base imbalance,*** provides assessment about ***the adequacy of oxygenation and ventilation.***

The normal values of ABG measurement:

- ***Oxygenation*** is assessed by PaO_2
- Normal value of PaO_2 is **80–100 mm Hg.**
- Hypoxemia is a state in which the PaO_2 is **< 60 mm Hg.**
- Hypoxia is a state in which there is inadequate O_2 at the tissue level.

Factors which modify the PaO_2:

- Age
- Altitude

$\boxed{\text{Both of which decrease the } PaO_2.}$

- Administration of supplemental oxygen.
- ***Oxygenation*** can further be assessed by SaO_2 (SpO_2)
- Normal value is, **92%–100%**
- At 92% saturation, the PaO2 is approximately 60 mm Hg.
- Lower values indicate Hypoxemia. (At this point, a recollection of the orientation of Oxygen Dissociation Curve may be needed)
- ***Ventilation*** is assessed by $PaCO_2$
  - Normal value is, **35 mm–45 mm Hg**
- ***Acid-base status*** is assessed by, **pH, $PaCO_2$, HCO_3,** and **Base Excess.**

The Base Excess (BE) reflects an increase or decrease in the total buffer base. It is an indication of metabolic make up of acid base disturbance.

- A decrease in BE – indicates loss of base, so ***Metabolic Acidosis.***
- An increase in BE – indicates addition of total base, so ***Metabolic Alkalosis.***

Normal ABG values		*Variations*		
		Acidaemia	*Alkalaemia*	*Hypoxaemia*
pH:	**7.35–7.45**			
PaO_2:	**80–100 mm Hg**			Mild: <80 mm Hg Moderate:<70 mm Hg Severe: < 60 mm Hg
$PaCO_2$:	**35–45 mm Hg**			
HCO_3^-:	**22–26 mEq/L**			
BE:	**– 2 to + 2 mEq/L**			
SaO_2:	**92% to 100%**			< 92%

STEPS FOR ANALYSIS OF ABG VALUES

There are five steps for analysis of ABG values:

- Look at each number individually; decide whether the value is **High, Low,** or **Normal,** and label the finding.

For example, a pH value of 7.50 would be high and labeled as alkalaemia.

- Describe the adequacy of **oxygenation** by assessing **PaO_2** and **SaO_2.**
- Determine the acid base status by assessing **the pH.**
- Decide whether the acid-base disorder is **respiratory** or **metabolic**. Check the PaO_2 (respiratory) and the HCO^- (metabolic) to see which one is altering in the same manner as **the pH.**
- Use the base excess to confirm the interpretation, to see **Respiratory, Metabolic,** or **Mixed,** especially when the disorder is mixed. In a mixed disturbance there is acid base imbalance in both the systems.
- Determine the **extent of compensation.**
  - Look at the system (respiratory or metabolic) that does not match the pH to determine whether it is moving out of its normal range in an effort to correct the acid base disturbance.
  - ***Absent:*** The value of opposite system is normal, indicating that no compensation is occurring. The pH is assumed to be abnormal.
  - ***Partial:*** If the value that does not match the pH status and is above or below the normal range and the pH is still outside the normal range, then partial compensation has occurred.
  - ***Complete:*** The value that does not match the pH and is above or below normal, but the pH is normal.

Examples

I

pH:	**7.34**	(Acidemia)
PaO_2:	**129**	(Adequate oxygenation)
$PaCO_2$:	**48**	(Acidemia)

HCO_3^-:	**26**	(Normal)
BE:	**+1**	(Normal)
SaO_2:	**99%**	(Normal)

Interpretation: Respiratory acidosis with no compensation, adequate oxygenation.

II

Patient with chronic bronchitis, emphysema and cor pulmonale, treated with digitalis and diuretics.

pH:	**7.4**	(Normal)
PaO_2:	**57**	(Hypoxemia)
$PaCO_2$:	**58**	(Acidemia)
HCO_3^-:	**26**	(Normal)
BE:	**+9**	(Alkalemia; use to determine whether the primary disorder is respiratory or metabolic.)
SaO_2:	**89%**	(Hypoxemia)

Interpretation: Metabolic alkalosis with complete respiratory compensation, hypoxemia.

III

Sixty two year old man with history of cancer. Status post-abdominal surgery for drainage of abscess.

pH:	**7.29**	(Acidemia)
PaO_2:	**192**	(Normal)
$PaCO_2$:	**40**	(Normal)
HCO_3^-:	**19**	(Acidemia)
BE:	**– 5.6**	(Acidemia)
SaO_2:	**97.5%**	(Normal)

Interpretation: Metabolic acidosis with no compensation, adequate oxygenation.

IV

Thirty-four years old man with 85% total body surface area burns in a house fire. He initially presented with a pH of

7.18. He was resuscitated with 40 L Lactated Ringer's solution and 6 ampoules of $NaHCO_3^-$. The patient is sedated, medically paralysed, and being mechanically ventilated with a V_E of 17 L/min.

pH:	**7.37**	(Normal)
PaO_2:	**126**	(Normal)
$PaCO_2$;	**40**	(Normal)
HCO_3^-:	**20**	(Acidemia)
BE:	**– 3.1**	(Acidemia)
SaO_2:	**98%**	(Normal)

Interpretation: Fully compensated metabolic acidosis with adequate oxygenation.

Interpreting ABG in Terms of V/Q Mismatches

- Abnormalities in V/Q (Ventilation-Perfusion) matching are evident in the patient's ABG values.
- Low V/Q units, shunts, etc. cause hypoxaemia whereas high V/Q units, dead space, etc. result in a rising $PaCO_2$.
- The patient's clinical presentation also provides significant information regarding the possible presence of a V/Q mismatch.
- The patient who initially has an increasing V_E (Minute ventilation) and a rising $PaCO_2$ may have a hyper-metabolic or other state resulting in CO_2 production that exceeds the patient's ventilatory capability or they may have increasing dead space.
- When the dead space increases, the ventilatory demand increases. The concern for the clinician is what is causing the increased V_D/V_T and whether the patient has sufficient reserve to meet the increased demand.

Example:

Two patient with increased V_E as a result of increased V_D/V_T.

	Compensated patient	*Uncompensated patient.*
V_E:	18 L	16 L
$PaCO_2$:	40 mm Hg	55 mm Hg
pH:	7.40	7.33

Discussion

- Out of the two, only one patient is able to maintain acid base balance.
- Both the patients are at risk of developing fatigue and ventilatory failure because of high demands placed on the systems and need to be monitored carefully.
- However, the uncompensated patient is at higher risk of developing ventilatory failure and likely need for intubation and mechanical ventilation.
- The underlying mechanism resulting in increased V_D/V_T (e.g. Pulmonary embolus, decreased cardiac output, etc) must be identified and appropriate treatment must be instituted.
- Pulmonary embolism is treated with low dose heparin, thrombolytic therapy, etc.
- Decreased pulmonary perfusion due to low cardiac output need therapy towards optimizing the hemodynamic status.
- *Alveolar overdistension caused by overzealous mechanical ventilation is managed by adjustment of ventilator settings.* Changes may include decreasing the V_T (tidal volume), changing the flow pattern or mode in an attempt to *reduce the inspiratory pressure.*

The patient who has hypoxemia despite supplemental oxygen likely has *increasing shunt.* Many therapies may be instituted in an effort to decrease shunt.

- It has to be remembered that shunt is the most common cause of hypoxemia in intensive care units.

- Pulmonary hygiene interventions range from cough and deep breathing to therapeutic bronchoscopy.
- Pulmonary edema may be managed partly with diuretics.
- Bronchospasm is managed with bronchodilators, both systemic as well as topical inhalation of aerosol therapy.
- If mechanical ventilation is used, PEEP may be used to re-expand the alveoli and decrease the physiologic shunt (recruiting more of the alveoli).
- It must be always remembered that the ventilator serves as a mechanism to support oxygenation and ventilation, *not as a primary treatment for ventilation/perfusion abnormalities.*
- Identification and management of the underlying pathology and application of physiologic measures to monitor patient's progress, and dynamic revision of the plan of care are absolutely essential for bringing the patient back to normal.

Technique of Obtaining Arterial Blood Sample

- The radial artery is the common site for taking an arterial blood sample for analysis.
- The other sites are brachial artery and femoral artery in adults.
- Femoral artery is commonly chosen for emergency use, as it is easy to reach, particularly when the patient is in shock with feeble pulse.
- Before doing an arterial puncture, assessment of adequate collateral circulation is made to be sure that if a hematoma is formed it does not compromise the circulation.

- *Modified Allen test* is done in hand for this purpose. This is done by elevating the hand and compressing the radial and ulnar arteries and the patient is asked to close and open the fist till the hand blanches. Now releasing the pressure from ulnar artery and assessing the return of normal colour. An erythematous blush is a positive Allen test result whereas continuing pallor indicates poor collateral circulation. Therefore, arterial puncture is not done at that site.
- Coagulopathy or medium to high dose anticoagulation therapy is a relative contraindication for arterial puncture. If only a single puncture is needed, adequate time of compression (at least 5 minutes) is allowed to achieve hemostasis.
- Repeated puncture of a single site is avoided to prevent hematoma, scarring, laceration of artery, etc.
- When continuous invasive blood pressure monitoring and arterial blood gas monitoring are needed, cannulation of artery is necessary. Patency of arterial catheter is achieved by a continuous flush device. All connections of this line must be carefully secured otherwise loose connections may result in rapid blood loss.
- When the sample of blood is taken, it is carefully handled to prevent inaccurate results. For prevention of clotting, liquid or powder heparin is added to the syringe. The barrel of the syringe must be just wet and the excess heparin must be flushed out, otherwise the sample will be acidified by the heparin, as the pH of heparin is 7.0.
- Samples must be taken anaerobically and any air bubble should be expelled rapidly. Presence of air will increase oxygen level and lower the CO_2 level.

- The sample must be kept immediately in an ice bath to slow down metabolism. Otherwise oxygen level will be lowered and CO_2 level will be raised.
- If the sample is kept at room temperature, the analysis must be performed within 15 minutes. If it is kept at 4°C, analysis can be done within an hour.
- Patient's body temperature at the time of collecting the sample must be noted in the requisition, as the values are given, assuming the body temp as 37°C.
- Temperature above 37° C shows higher value of O_2 and CO_2 values, whereas the temperature below 37°C will have the opposite effect. That is because of the changes in the oxygen dissociation curve.

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CHAPTER 11

Indications for Mechanical Ventilation and Respiratory Failure

❖ *Indications for ventilator therapy*
❖ *Respiratory failure*
❖ *Two types of respiratory failure*

Mechanical ventilation is an invasive therapeutic procedure with its own indications, merits, demerits, limitations, and dangers.

In the care of many acutely ill patients, mechanical ventilation lasting only hours or a few days is sufficient and the ventilator can be removed and normal breathing resumes. Unfortunately, for those whose underlying disease is chronic or irreversible, can become, sometimes unexpectedly, chronically ventilator dependent. Their continuing need for mechanical ventilation may be total, i.e. 24 hours a day, or it may be limited, i.e. only during sleep or intermittently through the day.

It must be remembered that the application of mechanical ventilation does not correct the underlying disorder. It only *supports* the respiratory system until the appropriate therapies cure the underlying disorder.

- It is frequently referred to as "ventilator support".
- Like any other support, it has to be given for the shortest duration possible.
- Any treatment has many indications.
- If applied for a proper indication in appropriate manner it leads to a successful out come.

It is very apt at this moment, to recollect the famous quotation by Sir Robert Macintosh;

"The drug is not dangerous, but the man who administers it is"

—Sir Robert Macintosh.

Therefore, identifying the indications for mechanical ventilation by clinical parameters and judgment is absolutely essential. Once it is recognized, it may be established by other indices and investigations and ventilator support may be instituted.

- Some ***clinical indications*** for mechanical ventilation:
  - Impending apnea
  - Paradoxical respiration
  - Obvious fatigue
  - Persistent tachypnea
  - Variable respiratory rate
  - Absent protective reflexes
  - Threat of airway obstruction

The primary indication for mechanical ventilation is impending or established Respiratory Failure.

Hence, in this chapter the indications for mechanical ventilation are discussed in detail.

There are two terms which need special concern; namely *"The ventilatory supply"* and *"the ventilatory demand"*.

- ***Ventilatory supply*** *is the maximal sustainable ventilation that a person can uphold without respiratory muscle fatigue.*
- ***Ventilatory demand*** *is the minute ventilation required to maintain a normal baseline* $PaCO_2$.

In simpler terms, ventilator support is needed in any situation where ventilatory demand exceeds the ventilatory supply.

The decision to intubate and mechanically ventilate a patient is based on sound clinical decision making after assessment of patient's *"Oxygenation"*, *"Ventilation"*, and *"Work of breathing"*. This decision is not arbitrary, because of the complications and problems associated with artificial airways and positive pressure ventilation.

However, the decision to initiate mechanical ventilation is a clinical one, but certain parameters have been suggested as guidelines.

RESPIRATORY GAS TENSIONS

Direct Indices

- Arterial oxygen tension: < 50 mm Hg on room air
- Arterial CO_2 tension: > 50 mm Hg in the absence of metabolic alkalosis

Derived Indices

- PaO_2/F_IO_2 ratio: < 300 mm Hg
- $P(A - a)O_2$ gradient: > 350 mm Hg
- V_{D}/V_T: > 0.6

Clinical Indices

- Respiratory rate: > 35 breaths/min.

Mechanical Indices

- Tidal volume: < 5 ml/kg
- Vital capacity: < 15 ml/kg
- Maximum inspiratory: force < – 25 cm H_2O

The need to institute IPPV is sometimes straight forward, as in the sudden onset of apnea in a patient with head injury. However, more frequently the situation is not clear cut.

- A balance has to be struck between the clinical situation, the symptoms and signs, and the arterial blood gas.

- If there is any doubt, initial treatment with high flow oxygen therapy by face mask, preferably with a reservoir device to maximize F_IO_2.
- The treatment is guided by repeated measurements of arterial blood gas, and provided oxygen therapy is correctly prescribed, humidified, and monitored; the dangers are minimal.
- The theoretical danger of inducing respiratory depression in patients with CO_2 retention has been overemphasized.
- If the decision not to ventilate is made in equivocal case, but the patient must be observed closely in a high care area such as a resuscitation room or anesthetic recovery room or a high dependency unit.
- The use of peripheral pulse oximeter is indispensable, *but may not warn of dangerous hypoventilation if supplemental inspired O_2 is given.*
- Patients with neuromuscular disorders, especially bulbar palsy or diaphragmatic weakness, may not be able to cough adequately to clear secretions, tracheal intubation may be necessary for bronchial toileting.
- Hypoxia refractory to high-flow inspired oxygen, with signs of respiratory distress, is a strong indication for mechanical ventilation.
- Some ***biochemical indications*** for mechanical ventilation:
  - PaO_2: < 60 mm Hg on high F_IO_2
  - Pulse oximeter: $< 90\%$ on high F_IO_2
  - $PaCO_2$: > 53 mm Hg or rising from a lower level
  - $PaCO_2$: persistently < 26 mm Hg

POSTOPERATIVE ARTIFICIAL VENTILATION

Who Should Receive?

- Patients unable to maintain adequate levels of oxygenation or who develop hypercapnea.
- Patients with pulmonary pathology which is potentially reversible.
- Inability to maintain a satisfactory $PaCO_2$.
- Blood gas values may be normal but the patient is exhausted (Over working).

Initial treatment of hypoxemia is by administration of O_2 by face mask (Using a fixed performance mask). At least 40% of oxygen must be administered.

- The dangers of oxygen therapy are over estimated and in the ICU environment, where a skilled nurse is attending the patient all the times, O_2 should be administered in a concentration which achieves satisfactory O_2 saturation.
- In the meantime the effect oxygen therapy must be closely monitored by pulse oximetry and blood gas analysis carried out after 25 to 30 minutes. This will give more reliable measure of oxygenation as well as providing information about PCO_2 and acid base status.

Pain

- If pain rather than weakness is the major defect, may often be managed conservatively, if first class pain relief is provided. For example, epidural analgesics or opioids or IV infusion of opioids, etc.

Exhaustion

- Exhaustion is indicated by laboured pattern of rapid shallow breathing which is often accompanied by deterioration of level of consciousness.

- Mechanical ventilation may probably be required if the respiratory rate remains at or above 45/min for more than an hour.
- It has been established in a study that "80% of post-operative deaths occur in the first postoperative hour, out of which more than 75% die of hypoventilation caused by recurarisation due to residual NMB.

Respiratory Failure

During normal respiration, the individual's maximum sustainable ventilation (**Ventilatory supply**) is very much greater than the individual's spontaneous minute ventilation (VE) (**Ventilatory demand**). Hence, the ventilatory reserve is very good that even when there is an increased demand as in a severe exercise, supply meets the demand, and there is no failure.

The plain fact is that when ***the ventilatory demand*** of the patient exceeds ***the ventilatory supply,*** then inevitably ***respiratory failure*** is caused.

It may be due to either ***the demand is increased*** or ***the supply is reduced.***

Both the situations will lead to a clinical condition called ***respiratory failure*** which manifest by various indices discussed above.

Therefore, plainly ***a patient in respiratory failure needs ventilatory support (Mechanical ventilation).*** Hence, it is necessary to discuss in detail about the various mechanisms that produce respiratory failure and how it can be supported by mechanical ventilation.

RESPIRATORY FAILURE

Definition

- It is defined by Campbell, *as a state present in a patient at rest, breathing air at sea level, because of impaired*

respiratory function, ***the arterial PO_2 is below 60 mm Hg*** or ***the PCO_2 is above 49 mm Hg*** (Campbell).

- "It is simply, the inability of the lungs to produce adequate arterial oxygenation, with or without acceptable elimination of CO_2." This may be acute or chronic.

Whether this definition explains everything well is questioned.

A variety of primary or secondary *disorders of airway, lung parenchyma, chest wall, and neural process of breathing* may be responsible for respiratory failure.

Common Causes of Respiratory Failure

Causes of Failure to Maintain Normal Blood Gas Homeostasis

Respiratory depression:

It can be due to either of the following two,

- Central depression by opioids or other depressant drugs.
- Depression of peripheral neural mechanisms as in Guillain Barre Syndrome or Myasthenia Gravis or clinically simply by neuromuscular blocking drugs.

Respiratory obstruction:

- Obstruction may be anywhere in the respiratory tract or alveoli starting from upper airways down to the alveolar capillary membrane.

Pulmonary failure:

It may include,

- Chronic bronchitis, acute bronchiolitis, Emphysema, Fibrosis, Asthma, ARDS, Pneumonia, Pneumothorax, and Pulmonary Edema.

Cardiac failure:

- With resultant pulmonary congestion interstitial edema.

Upper abdominal surgery:

This may result in,

- A restrictive syndrome immediately after surgery, with up to 60% reduction in FEV_1 and FVC. It may recover in a week's time.
- Changes in the pattern of breathing, i.e. rapid and shallow breathing sometimes persisting even after complete pain relief with opioids.
- A reduction in FRC starting during surgery, maximal at 24 hours and resolving in a weeks time.
- Insufficiency in diaphragmatic function.

Thoracic Surgery:

- Diffuse atelectasis and reduction in diaphragmatic function.
- Phrenic nerve and other injuries during cardiac surgeries.

Acidosis: Acidosis of any etiology

Hypermetabolic states:

- Hyperpyrexia, Thyroid crisis, etc.

Respiratory muscle dysfunction:

- Respiratory muscle fatigue or paralysis is the most common factor precipitating respiratory failure.

In general terms,

- The inadequacy in accomplishing movement of air in and out of lung is called as *Ventilatory failure.*
- The inadequacy in accomplishing gaseous exchange between *the alveoli* and *blood* stream is called as *Respiratory failure.*

- However, in clinically, a common term *Respiratory Failure* is used.

How far is it scientifically correct to use the term ***Respiratory Failure?*** It can be analysed, by going step by step.

Physiologically respiration is some times referred to as ventilation. ***Respiratory insufficiency*** is referred to as ***Ventilatory insufficiency***. These terminologies should not cause any confusion in the minds of readers, particularly when dealing with the mechanical ventilation. Are these two words, respiration, and ventilation one and the same?

Basic Mechanism of Respiration

It is absolutely essential to recall the basic components of respiration (Fig. 11.1).

- Respiration is divided into two parts; **External** respiration and **Internal** respiration.
- **Ventilation is** movement of air into and out of the lungs.

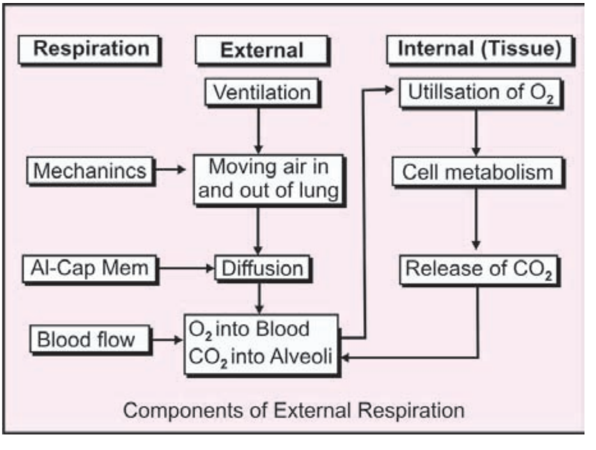

Fig. 11.1: External and internal respiration with their components

- **Respiration** is the movement of gases across a membrane.
- **External respiration** is the movement of oxygen and carbon-dioxide between the alveoli and the blood stream (diffusion).
- **Internal respiration** happens at the cellular level.

Having discussed all these a more refined definition may be arrived at.

Therefore, respiratory failure is,

"Absence of normal homeostatic state of ventilation as it relates to the acid-base status of blood and the exchange of oxygen and carbon-dioxide." This defect is reflected in the patient's blood gases.

Objective clinical definition of respiratory failure can be:

- A PaO_2 of < 60 mm Hg on an F_IO_2 of > 0.5 (relates to **Oxygenation**)
- A $PaCO_2$ greater than 50 mm Hg, with a pH of 7. 25 or less (related to **Ventilation**)

Hence, the term ***"Respiratory failure"***, is absolutely correct to be used, whether the patient's blood gas homeostasis is altered due to a defect in *ventilation* or in *oxygenation* since ultimately delivery of oxygen to tissue is affected.

The differentiation is,

- If it is due to inadequacy of ventilation, it is ***Hypercapnic*** *respiratory Failure.*
- If it is due to inadequacy of oxygenation, it is ***Hypoxemic*** *respiratory Failure.*

Obviously in both the conditions support is required to establish normalcy and the type of support may be different.

Clinically the patient may be in respiratory failure due to any of the following reasons,

- Failure to ***Ventilate.***
- Failure to ***Oxygenate.***
- ***Combination of*** failure to ventilate and failure to oxygenate.

Two types of respiratory failure

Respiratory Failure	
Hypercapnic failure (Respiratory pump failure)	*Hypoxemic failure (Lung failure)*
1. Central Nervous System **2.** Peripheral Nervous System and Respiratory Apparatus (Chest Bellows) **3.** Airways	**1.** Alveolar (Alveoli and Alveolar-Capillary Membrane)

Clinical observation of patient may give some clue that respiratory failure is present (Fig. 11.2). They may be,

- *Restlessness* due to impending cerebral hypoxia and fatigue.
- *Fatigue* due to manifold increased work of breathing.
- *Sweating* due to increased sympathetic activity .
- *Cyanosis* due to severe fall in O_2 saturation (falls in SPO_2 and PaO_2/F_IO_2 ratio).
- *Tachycardia.*
- *Ectopic beats.*
- Complaints of *Dyspnea.*
- *An increasing respiratory rate.*
- *A decreasing tidal volume.*
- An increase in the work of breathing as evidenced by the *use of accessory muscles of breathing.*
- *Tracheal tug.*
- Reduction in the number of words per breath.
- *Paradoxical breathing.*

Hypercapnic failure is likely to occur because of the inability to sustain adequate ventilation by one or more

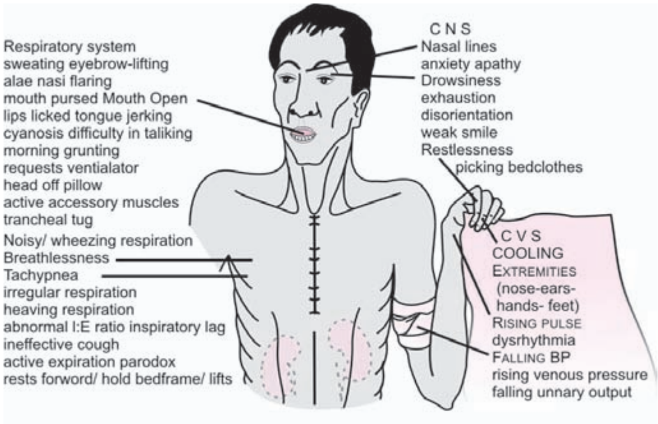

Fig. 11.2: Some of the manifestations of hypoxia in a patient with respiratory failure

components of ventilation namely the central nervous system (respiratory centers), the peripheral nervous system innervating the respiratory muscles, and the airways.

Hypoxemic failure occurs out of inability to maintain adequate exchange of gases at the alveolar level (Alveolar capillary membrane).

Failure to ventilate (This is also known as ventilatory failure, hypercapnic failure or respiratory pump failure) is caused when ventilatory demand exceeds ventilatory supply.

Ventilatory demand is the minute ventilation required to maintain a normal $PaCO_2$.

Ventilatory supply is the maximum sustainable ventilation which a person can maintain without any respiratory muscle fatigue.

For maintaining adequate gas exchange, the Ventilatory **supply** (the individual's maximum sustainable ventilation)

must meet or exceed the Ventilatory **demand** (the individual's spontaneous minute ventilation to maintain a normal $PaCO_2$:

- In normal individuals—the **supply** is very much **greater** than the demand (Fig. 11.3).
- In borderline ventilatory failure—the **supply** approximately **equals** the demand (Fig. 11.4).
- In established ventilatory failure—the **supply** is significantly **smaller** than the demand (Fig. 11.5). (The demand exceeds the supply).

When the demand exceeds the supply Mechanical Ventilatory support is necessary.

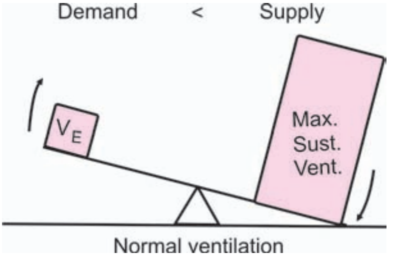

Fig. 11.3: Ventilation in normal individual

- ❖ Here the maximal sustainable ventilation exceeds the minute ventilation.

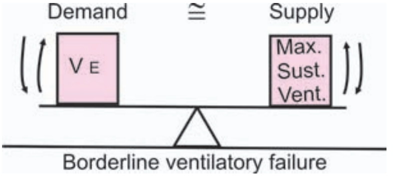

Fig. 11.4: Ventilation in borderline ventilatory failure

- ❖ The maximal sustainable ventilation almost equals the minute ventilation.
- ❖ A balance is maintained with a lot of compromise.
- ❖ This situation arises because of an increase in ventilatory demand or decrease in ventilatory supply or both.

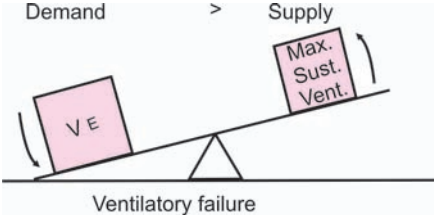

Fig. 11.5: In ventilatory failure

- When the demand exceeds the supply, *respiratory failure* is established.
- In this situation *mechanical ventilatory support* becomes necessary.

Two types of respiratory failure have to be understood. It has to be clearly differentiated which type of respiratory failure the patient suffers from. On the other hand, if he has a combination of the two types it has to be identified. Based on the cause, appropriate therapeutic decision has to be made.

Failure to Oxygenate

The primary cause of failure to oxygenate is **"Ventilation–Perfusion mismatch**", where ventilated oxygen is not interfacing with the pulmonary capillaries and so adequate oxygen does not reach the hemoglobin. This situation creates a persistence of hypoxemia. Conservative measures such as supplementation of additional oxygen in the inspired air may correct it; however if it is not corrected, then quick decision to intubate and mechanically ventilating the patient is mandatory to prevent tissue hypoxia

Failure to Ventilate

Failure to ventilate is clinically different from ***failure to oxygenate***, as ventilatory failure is related to failure of the

mechanics of respiration resulting in inadequate movement of air in and out of lungs leading to failure in maintaining normal arterial O_2 saturation and CO_2 elimination. Here again, conservative measure of supplementing inspired oxygen may improve the situation, if it is not corrected, ventilatory support will be indicated.

Ventilatory therapy will be very helpful, when the defect is with,

- Mechanics of breathing as in Guillain-Barrie syndrome (Acute ascending polyneuritis) or reversible blockade of neuromuscular junctions as in the case of inadequately reversed patients in postoperative period, certain envenomations like a cobra bite, etc.
- Certain conditions where the central control of breathing (respiratory centers) is depressed as in the case of opioid overdosage, or poisoning with central depressant drugs.
- In defects of parenchyma, it may be difficult to establish adequate gas exchange is spite of ventilatory support and depending upon the type of damage, modified modes may improve the gaseous exchange.

Mechanical ventilation allows the following merits.

- Support of an increased ventilatory demand (by increasing the supply).
- Application of tidal volumes that will increase the Functional Residual Capacity (FRC).
- Improve oxygenation by increased inspired oxygen concentration and application of Positive End Expiratory Pressure (PEEP).

The decision to intubate and ventilate the patient with a mechanical ventilator is critical, as intubation and positive pressure ventilation are not innocuous procedure and free from dangers.

It is possible a wide variety of diseases affecting the Central Nervous System, Neuromuscular functions, musculoskeletal system, Conducting Airways, and the gas exchanging units (Alveolar Capillary membrane) may present with respiratory failure. Suitable therapy directed towards the management of the primary problem is always the sheet anchor.

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CHAPTER 12

Maintenance of Airway and Tracheal Intubation

- *The common causes of upper airway obstruction*
- *Different artificial airways for protecting patient's airway*
- *Endotracheal intubation*
- *Indications for endotracheal intubation*
- *Laryngoscopes*
- *Technique of intubation*
- *Stabilisation of endotracheal tube*
- *Different types of endotracheal tubes and cuffs*

Nothing can be done to reverse hypoxic brain damage once it occurs. Because of this fact, maintaining an airway and ensuring adequate oxygenation supersedes everything else. There is an old medical saying; "Patients do not die or suffer brain damage because you cannot intubate them; they die or suffer brain damage because you can not oxygenate and ventilate them."

Therefore, the most vital requirement for adequate respiration is a patent and clear airway. So also when a patient is in respiratory inadequacy, the first thing to be checked is the patency of airway and if necessary, to take steps to maintain the patency.

Hence, before going into the details of actual ventilatory support, it is essential to look into the basic steps for maintaining a clear and patent airway so that the available movement of air into the lungs is not blocked at that level.

- The upper airway is the commonest site of obstruction in a critically ill patient or unconscious person.
- If the upper airway patency is maintained, it will be possible to assess whether the available ventilation is adequate or not.
- Some times, the patient may be seen with a borderline hypoventilation with upper airway obstruction. In this situation, clearing the airway obstruction may simply

solve the problem, as seen by the improvement in oxygen saturation. In spite of maintaining the airway normal, if the saturation does not improve, a modest supplementation of oxygen may solve the problem. If oxygen saturation improves with supplementary oxygen, it can be reasonably presumed that the CO_2 elimination would be near normal.

However, when there is difficulty in maintaining the airway, artificial airway may be used to establish patency and control of the airway. Careful monitoring of the airway and use of an artificial airway may be indicated when,

Partial or complete airway obstruction is seen.

For preventing aspiration, when the protective reflexes are inadequate or absent.

For removal of secretions from the respiratory tract.

For providing mechanical ventilation.

Maintenance of airway in an unconscious patient.

THE COMMON CAUSES OF UPPER AIRWAY OBSTRUCTION

- Foreign body like secretions, food, vomitus, and blood clots.
- Expanding hematoma or edema.
- Depressed level of consciousness with loss of muscle tone.

Manifestations

- Increased respiratory efforts.
- Classical sign in upper airway obstruction is “Stridor”, a shrill, crowing sound during inspiration.
- Total airway obstruction is an emergency.
- Patient may be extremely agitated, and holds the throat.

- May have a depressed level of consciousness.
- Work of breathing is enormously increased and supra sternal and intercostals indrawing may be noticed.

Airway management does not mean intubation. "It means just that, managing the patient's airway to ensure patency, provide adequate ventilation, and maintain appropriate oxygenation."

Many times, every one focuses on using advanced measures or procedures, forgetting that they are often useless or perhaps detrimental without the basis. Merely performing a chin lift, jaw thrust can open and/salvage many airways. Proper use of basic airway adjuncts such as oral or nasal airways, can convert a difficult – to ventilate patient into a stable, well-ventilated one.

Steps for maintaining the airway clear in an unconscious patient:

In fact many times the major catastrophe is caused by improper maintenance of the airway. Intubation of the trachea is the sure way of maintaining the clear airway. The following points are **highly significant and are to be remembered.**

- A patient with a clear airway breathes very effortlessly and there will be no noise during breathing.
- If the airway is ***partially obstructed***, there is usually ***noise*** during breathing.
- If the airway is ***completely obstructed***, then there is ***absolutely no sound.***
- *Very loud snore with apparently regular breathing also indicates respiratory obstruction.*
- Observation of movement of the patient's chest and abdomen invariably give an idea of cause of obstruction.
- The characteristic "paradoxical respiration" could be observed, where during inspiration as no air inters, the

chest is drawn in and abdomen protrudes out as the diaphragm contracts. The reverse occurs during expiration without any air movement.

- Quickly the patient becomes cyanosed. The airway has to be restored immediately to prevent hypoxic brain damage.
- *The obstruction in almost all cases is due to the relaxation of the muscles of neck and those support the mandible.* This causes the mandible with the tongue to sag backwards and the base of tongue sits on the posterior pharyngeal wall causing obstruction. This is commonly referred to as ***"falling back of tongue"*** (Fig. 12.1).

Fig. 12.1: The common mechanism of airway obstruction

- ❖ Relaxed mandible.
- ❖ Flexed neck.
- ❖ Total airway obstruction by the tongue sitting on the posterior pharyngeal wall.

- This can be very easily relieved by a simple maneuver which is a combination of two steps. First ***flexing the neck and then extending the head***. This is tilting the head backwards so that his nostrils point upwards. This lifts

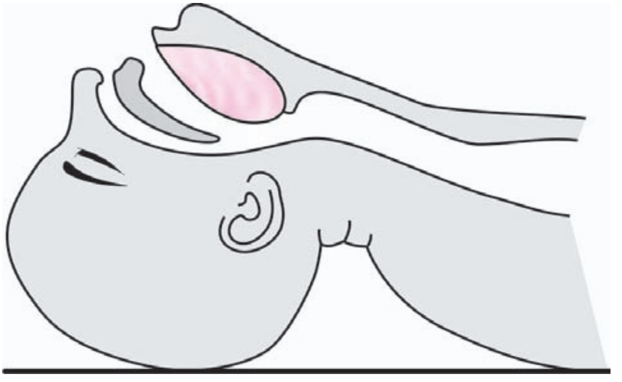

Fig. 12.2: Obstruction relieved
- ❖ Extension of head.
- ❖ Pushing the mandible upwards.
- ❖ Airway is clear.

the mandible and base of tongue away from the posterior pharyngeal wall and clears the airway (Fig. 12.2). (Head - tilt, Chin – lift and Jaw – thrust).

- When pulling the chin forward, ensure that the fingers are not jutted into the floor of the mouth under the chin, causing the tongue to be pushed farther back in the airway to cause obstruction.
- Head-tilt position is contraindicated in trauma patients for whom the stability of the cervical spine has not been ascertained.
- If this maneuver is not effective then pushing the jaw anteriorly by lifting at both the angles by the ring fingers of both hands and bringing the mandible to ***"prognathic" attitude***. This is keeping the lower incisors anterior to upper incisors. Position of the head and mandible for keeping the airway patent is shown in Figure 12.8).
- With these procedures usually the airway gets cleared and patient breathes normal. If there is no other

contra-indications to turn, the patient may be turned onto recovery position which takes care of airway and drainage of secretions. The patient may be retained in that position till he is able to maintain his airway well. (Fig. 12.3.)

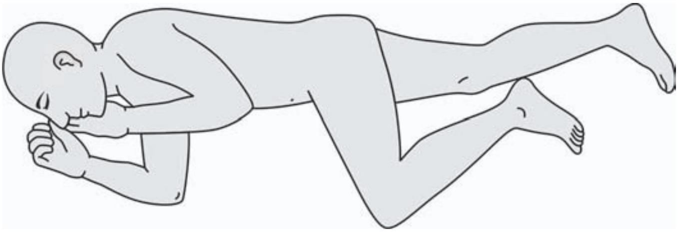

Fig. 12.3: Recovery position

- ❖ Patient is in a semi-prone position.
- ❖ Upper thigh is flexed to prevent him from falling on to the face.
- ❖ Head is turned to the side for draining the secretions.
- ❖ Head is slightly extended to keep the airway patent.

DIFFERENT ARTIFICIAL AIRWAYS FOR KEEPING PATIENT'S AIRWAY PATENT

Many airways are available for the maintenance of upper airway patency. Some of them are made of metal, some are of rubber and some are made of plastic. The common types are Waters airway, Guedel's airway, and Phillips airway (Fig. 12.4). The Guedel's airway is universally preferred as it is more anatomically shaped and is least traumatizing.

PLACING OROPHARYNGEAL AIRWAY IN POSITION (FIG. 12.9A)

- If properly placed, the airway holds the tongue away from the posterior pharyngeal wall. When in position it curves over the tongue, with its tip in the posterior pharynx.

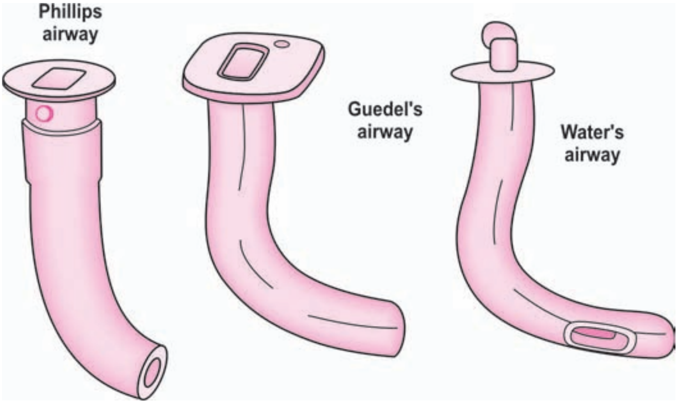

Fig. 12.4: Different airways

- ❖ Guedel's airway has many advantages.
- ❖ Anatomically shaped.
- ❖ Made of rubber or plastic.
- ❖ Less irritation to pharynx.

Technique:

- The proper size is absolutely essential and it can be assessed by placing the airway at the edge of the mouth, it should extend to the bottom of the ear) (Fig. 12.5).
- An airway that is too short will force the patient's tongue back into the pharynx thus cause airway obstruction.
- An airway that is too long will stimulate the gag reflex.
- This airway may be inserted "upside down" with the tip against hard palate the airway is slid into the mouth until the soft palate is reached, at which point it is rotated so that its curvature matches that of the tongue. The tip is advanced to the back of the mouth to ensure its position in the posterior portion of pharynx. The flange of the airway must rest on the teeth and the bite piece in between the incisors (Fig. 12.6).

Fig. 12.5: Assessing the size of airway

- Another way of insertion uses a tongue blade to depress the patient's tongue while inserting the airway, matching the curvature of the tongue.
- After the insertion of airway, the assessment of adequacy of ventilation must be done by observation and auscultation of breath sounds.

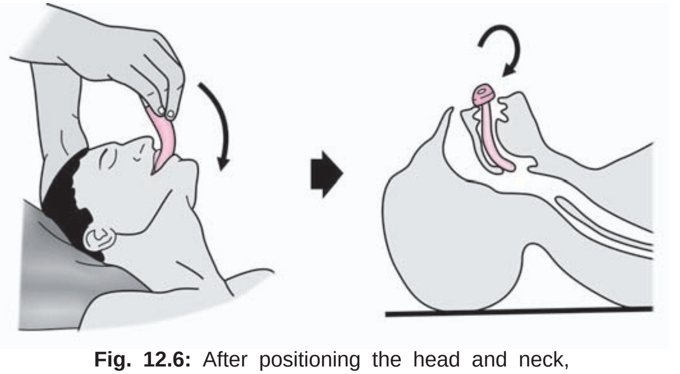

Fig. 12.6: After positioning the head and neck, a Guedel's airway is inserted to maintain patency of airway

If muscle power and reflexes have not recovered during this time, manually maintaining the airway like this may be difficult. Now the right size ***Guedel's airway*** may be inserted properly in the oral cavity and simple lifting of chin will maintain the airway. The different sizes of Guedel's Airway are shown in Figure 12.7.

- Sometimes the patient may not tolerate the oropharyngeal airway (Guedel's airway). Then forcing that in position may induce vomiting.
- Now a ***nasopharyngeal airway*** well-lubricated with local anesthetic jelly can be passed through the widest nasal cavity and left near the glottis by carefully listening to the breath sounds at the proximal end of the airway and fixed in that position.

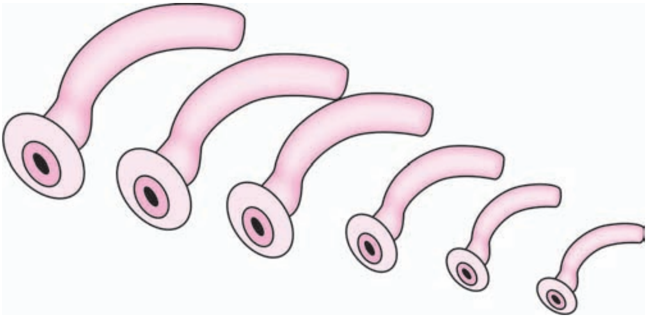

Fig. 12.7: Different sizes of Guedel's airway

Nasopharyngeal Airway

The nasopharyngeal airway is a soft tube made of rubber or latex with a bevel at the tip and a adjustable flange at the proximal end to stop it from slipping too much into the pharynx. This is available in plastic also.

- The appropriate size can be chosen by measuring the length from the nares to the tip of the ear. Too long an airway will enter into the esophagus. Too short will stop high in the nasopharynx or oropharynx and will not maintain a patent airway (Fig. 12.9B).
- Carefully ***clearing the oropharyngeal secretions by using suction*** with adequate power will keep things under control.
- If all these efforts fail to maintain the airway patent, the next option without causing further delay must be intubating the patient with a proper size cuffed endotracheal tube and observe him in recovery room till he is able to maintain his airway.

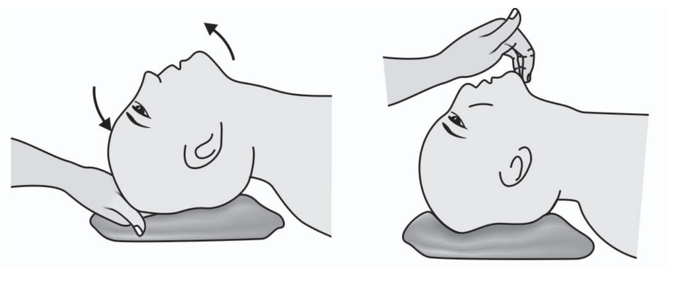

Fig. 12.8: Position of head and mandible for keeping the airway patent

- ❖ Positions of head and mandible for keeping the airway patent.
- ❖ Mandible is supported with fingers on the chin.

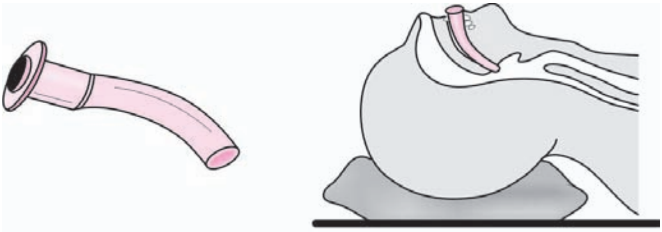

Fig. 12.9A: Keeping the airway patent by using oral airway

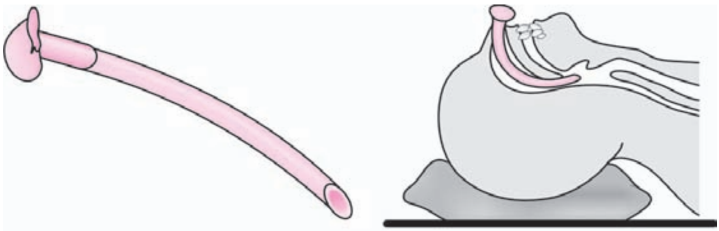

Fig. 12.9B: Keeping the airway patent by using a nasopheryngeal airway

❖ Maintaining the airway with nasopharyngeal airway.

ENDOTRACHEAL INTUBATION

Endotracheal intubation is accomplished by placing a tube known as "endotracheal tube" through the larynx into the trachea. The tube may be passed either through the nasal route or by oral route depending upon the necessity.

Indications for Endotracheal Intubation

1. Upper airway obstruction that persists in spite of conservative methods to maintain airway discussed already.
2. For secretion management, as the endotracheal tube provides a conduit for suctioning.
3. Protection of the tracheobronchial tree from aspiration of regurgitated material from stomach in patients with

a depressed level of consciousness or ineffective upper airway reflexes.

4. For supplementing higher concentration of oxygen, general anesthesia or mechanical ventilation.

Oral

- For maintaining a fool proof airway in any emergency situations.

Advantages

- Technically easy to do.
- Allows passage of a large tube; so less airway resistance to airflow.
- Removal of secretions is easier.
- Chances of kinking are less.

Disadvantages

- Less comfortable for the patient; may cause gagging, increase salivary secretions.
- Stabilization of the tube is relatively difficult.
- Patient may bite the tube causing a narrowing or total airway block.
- Does not allow oral hygiene maintenance.

Nasal

- For maintaining an airway in non emergency situations as elective intubation.
- For maintaining anaesthesia for oropharyngeal surgeries.
- Suspected patients with cervical spine injuries.

Advantages

- More comfortable for the patients; they tolerate the tube for longer periods.
- Less gagging.

- Easier to stabilize the tube and maintaining oral hygiene.

Disadvantages

- Technically more difficult to place the tube.
- Requires a relatively smaller tube to pass through the nasal cavity without trauma, so increased airway resistance.
- Kinks more easily.
- Epistaxis is possible during insertion.
- May contribute for sinusitis and otitis media.

Is tracheal intubation really too sophisticated and difficult technique? As a matter of fact it is one of the very easy techniques that can be learned and practiced without any difficulty. There can be hardly any more basic and life saving method in whole of medicine than inserting a tube into a patient's trachea to keep his airway open and also to protect his respiratory tract from any regurgitated food from stomach getting in and choking his airway. Many people strongly believe that intubation is anesthetist's job and they don't have anything to do about it.

Still more badly, many people believe that once a patient is intubated that is all about anesthesia. Both these ideas are totally incorrect.

Suitably trained assistants often intubate expertly, so this should surely be an essential skill for any doctor. In western countries the ambulance drivers and ambulance assistants are very well-trained in this technique, of course they are trained with the help of mannequins both in intubation and CPCR.

History

In 1788 C. Kite of Gravesend described oral and nasal intubation for resuscitation of the apparently drowned. Historically, tracheostomy was preferred to intubation because it was believed that a laryngeal tube will not be tolerated.

Edgar Stanley Rowbotham and Ivan Whiteside Magill first used endotracheal anesthesia by passing a gum elastic tube in trachea.

The first blind nasal intubation was performed by Stanley Rowbotham.

Magill published his results of blind nasal intubation with a wide bore rubber tube in 1929.

- Inflatable cuffs were used even earlier, but reintroduced in 1928 by Ralph Milton Waters and Arthur E. Guedel

Before the days of muscle relaxants, blind nasal intubation under deep inhalational anesthesia was practiced.

Use of muscle relaxant to facilitate intubation was pioneered by Bourne.

Endotracheal Tubes

Traditional tubes for either oral or nasal intubation were Magill tubes made of mineralized rubber to keep them retains their shape and lumen.

Relatively stiff without collapsing. The red color is due to the preservatives and to make it clear that it is for medical use. Oral tubes have thicker walls.

Their size is mentioned in mm, 8 mm, 8.5 mm, etc. which means that the internal diameter of the tube is 8 mm.

The red rubber tubes have cuffs made of soft latex rubber, when inflated become "fusiform" and the pressure inside the cuff is relatively high. (High pressure, Low volume cuff) (Fig. 12.19). When this pressure is higher than the perfusion pressure in the capillaries of submucosa of trachea, it may cause ischemic necrosis when used for a long duration.

Now disposable tubes made of semi rigid material, usually PVC (Poly vinyl chloride) or silicon rubber are available.

The toxicity of the PVC or other material is tested by implantation in rabbit muscle. Z79 is the committee in the US that approves anesthetic equipments. So in all disposable tubes the letters I.T. Z 79 would be printed to indicate that the material is tested by implantation test and is approved by the Z 79 committee.

All the materials red rubber, PVC, and silicon rubber are flammable.

The PVC tubes are relatively less irritant to the mucosa than red rubber tubes and hence may be used for many days provided it doesn't develop block of lumen.

These tubes come with soft "High volume Low pressure" cuffs that does not cause mucosal ischemia.

Different connectors are used to connect the endotracheal tube to the anesthetic breathing system or a resuscitator bag, etc.

Modern equipments use only the "Universal 15 mm connectors" available in all sizes stating from 2.2 mm tube meant for a premature neonate, so these tubes come with this type of connectors at the proximal end.

These connectors fit in almost all adapters used in equipments for anesthesia, resuscitation, as well as mechanical ventilation.

The body of the tube has a standard curvature, centimeter markings that allow for the determination of the depth of insertion, and radio-opaque markings either running through the length of the tube or at the distal end, so that the tube can be located on chest X-ray film.

The distal end of the tube has a beveled edge, which allows easier passage of the tube through the glottic opening.

A Magill type of endotracheal tube has an opening only at the distal end of the tube, whereas, a Murphy design tube has a small opening opposite to the beveled edge. This

hole allows ventilation if the bevel becomes lodged against the tracheal wall (Fig. 12.26).

Adult endotracheal tubes are provided with a cuff at the distal end which when inflated with air seals the trachea and allows application of positive pressure ventilation and minimizes the risk of aspiration.
The inflating system is a small bore tube fused within the wall of the endotracheal tube, emerging out at the proximal end in a pilot balloon which gives indication about the status of cuff after intubation (whether inflated or deflated).

This pilot balloon has a spring loaded valve to seal it deflating once the cuff is inflated with a syringe.

Laryngoscopes

A prototype was made by Chevalier Jackson (1865 – 1958) later it was modified by Magill, Paluel J. Flagg (1886 – 1970) of New York; Miller and Robert Macintosh of Oxford (1897 – 1989).

The light was originally powered from electric mains, but is now supplied from a 3 volt battery in the handle or by fibreoptic cable.

Magill's version is a straight blade with "U" shaped cross-section to lift the epiglottis.

Macintosh instrument is shorter, curved with "Z" shaped cross-section. Its tip enters the glossoepiglottic recess and lifts the base of the tongue and with it, the epiglottis, so that the vocal cords can be visualized. Most of the epiglottis is supplied by the internal branch of superior laryngeal nerve.

- In fact the introduction of muscle relaxants in clinical anesthesia in 1942 by Harold Randall Griffith (1894–1985) and G. Enid Johnson of Montréal, there was the necessity for ventilating a patient who is paralysed.

Moreover, a paralysed patient undergoing an abdominal surgery had to be protected from two dangers, namely inadequacy of ventilation and the danger of regurgitated material from the stomach getting aspirated into the lungs. In other words protecting the airway and maintaining the ventilation are the sheet anchors of a paralysed patient. This is easily done with the help of an endotracheal tube in trachea.

- The air tight seal of airway by an inflatable cuff was developed later.

After a minimal metamorphosis the right equipment designed by Sir Robert Reynolds Macintosh came into use in 1943. This is curved blade laryngoscope being almost universally used (Fig. 12.10). At least a few dozens of models were designed by various people and none stood the test of time as this design.

It is in use for more than 60 years and will be used for ever. It is the greatest credit to the inventor who had contributed enormous wealth in the form of equipment as well as research and writings to the field of anesthesiology (Fig. 12.11).

Fig. 12.10: Macintosh curved blade laryngoscope

Fig. 12.11: Macintosh laryngoscope with four blades

Laryngoscopy

- This is technique of *directly visualizing the laryngeal inlet with the help of the equipment known as Laryngoscope* usually for the purpose of intubating the trachea.
- This is relatively an easy technique that can be learned without difficulty from a good teacher still more easily particularly if a mannequin is available.

Technique of Intubation (Figs 12.12 to 12.27)

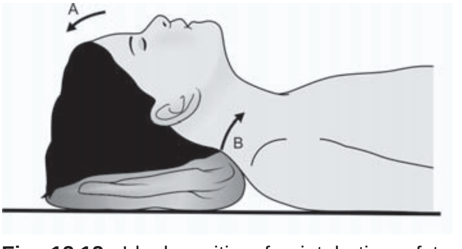

Fig. 12.12: Ideal position for intubation of trachea

- ❖ Keep a pillow under the head and neck, reaching up to the shoulder.
- ❖ Extension of atlanto-occipital joint, which makes the chin up.
- ❖ Flexion of the neck at the shoulder.
- ❖ Both these maneuvers will bring the airway in one line.
- ❖ Note that the shoulder is lifted by a pillow underneath for extending the head.

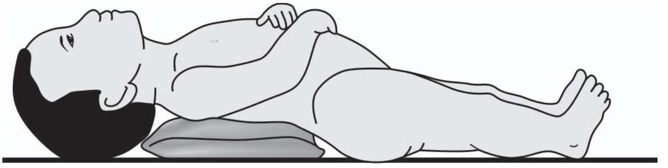

Fig. 12.13: Position of head and neck for intubation in a child

Fig. 12.14: Holding the laryngoscope in the proper way

Fig. 12.15: Introducing the laryngoscope

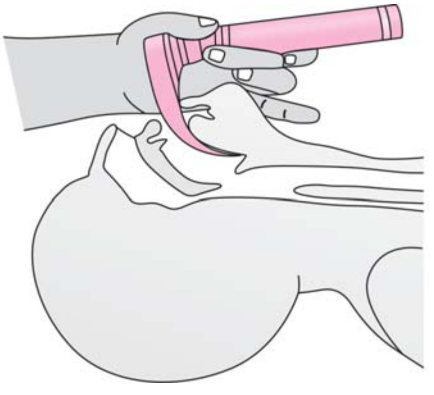

Fig. 12.16: Visualising the larynx

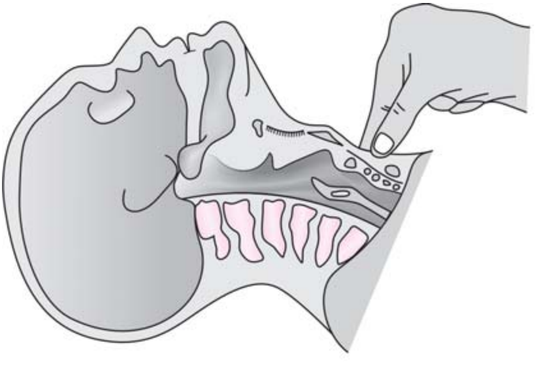

Fig. 12.17: Sellick's maneuver

- ❖ The pressure on the Cricoid ring pushes the larynx backwards to be visualised better.

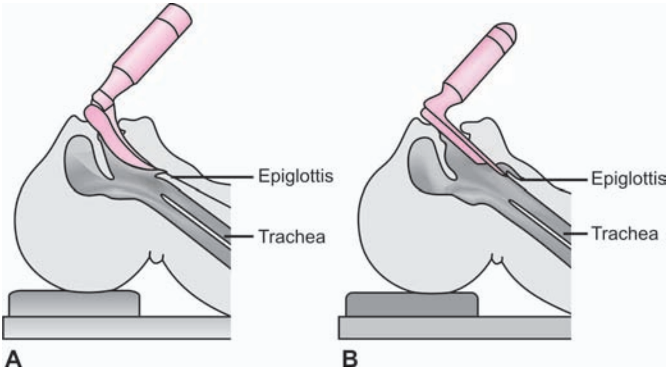

Fig. 12.18: Laryngoscopy

❖ Curved blade entering the glossoepiglottic fold.
❖ Straight blade lifting the epiglottis.

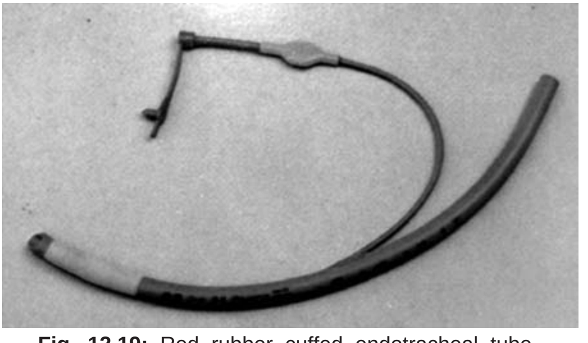

Fig. 12.19: Red rubber cuffed endotracheal tube

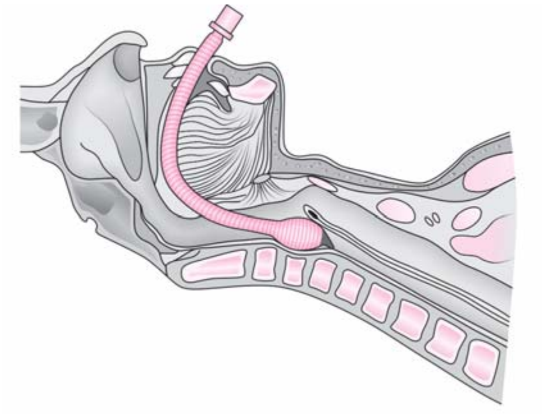

Fig. 12.20: Endotracheal tube in the esophagus

❖ Note the endotracheal tube is in the esophagus.

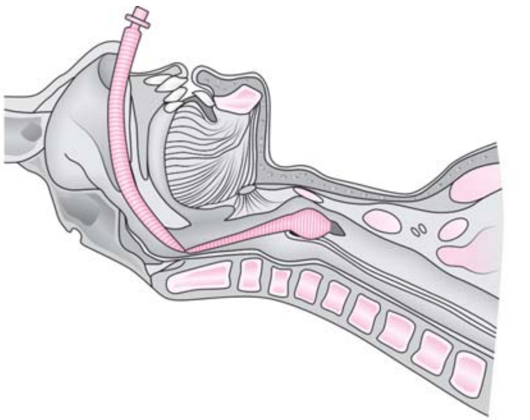

Fig. 12.21: Endotracheal tube getting kinked at the pharynx

❖ Note the kink at posterior pharyngeal wall resulting almost complete block.

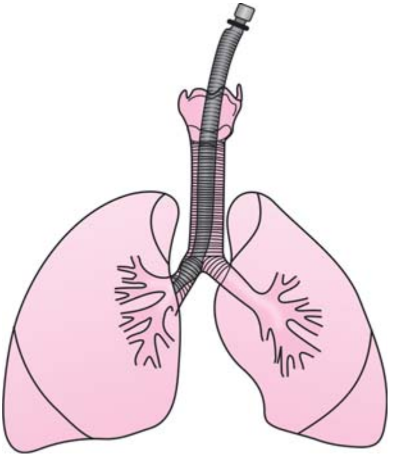

Fig. 12.22: Endotracheal tube in the right main bronchus

Fig. 12.23: Endotracheal tube with “Fusiform Cuff”

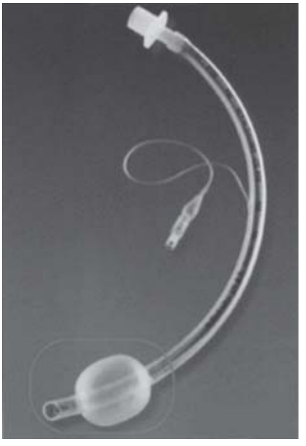

Fig. 12.24: Endotracheal tube with high volume—low pressure cuff

- ❖ This gives an air tight fit in the trachea without causing pressure on the tracheal mucosa and ischemic damage.

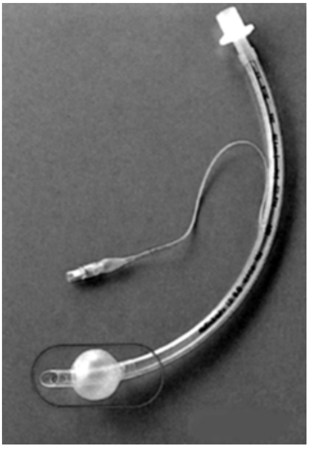

Fig. 12.25: Endotracheal tube with low pressure globular cuff

- ❖ The contact with the mucosa is less, so the seal is not very good.

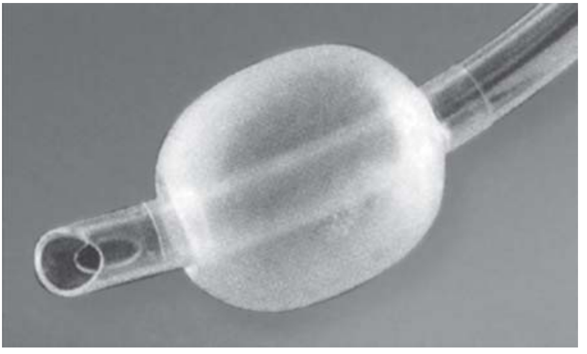

Fig. 12.26: An ideal high volume low pressure cuff

❖ Note the Murphy's safety eye on the tip of the tube.

Fig. 12.27: Anatomical face mask for ventilating the patient with 100% oxygen

❖ The brim is provided with and inflatable air cushion for better fit on the face.

INTUBATION

Intubation is the technique of passing a specially designed tube made either of red rubber or PVC of appropriate size into the trachea of the patient.

The Purpose of Intubation

- To maintain the airway without any obstruction in unconscious patients. This is otherwise difficult because of position, anatomy, surgery, etc.
- In unconscious patients to isolate the tracheobronchial tree with a proper size tube with a well fitting cuff and to protect it from getting soiled by aspiration of secretions or regurgitated material from the stomach.
- To apply tracheobronchial suction where there is collection of secretion that blocks the airway.
- During anesthesia, to deliver the anesthetic mixture into the tracheobronchial tree from where it reaches the alveoli. By diffusing through the alveolar capillary membrane it enters blood stream, reaches brain and causes anesthesia.
- In controlled ventilation anesthesia, the patient is paralysed by using muscle relaxants where his ventilation has to be taken over by the anesthetist till the muscle relaxant effect is reversed at the conclusion of anesthesia.
- To artificially ventilate patients who are in ventilatory insufficiency or failure. That is known as IPPV or ventilatory support.
- To reduce the dead space in patients with compromised ventilatory function.
- The term "Difficult intubation" is a vague one. It refers to the relationship between the skill of the intubator and the problems presented by the patient. Ideally the level

of competence matches the patient's needs – so the intubation is easy.

Human Assistance

Even in the most sophisticated environment, human assistance is absolutely essential when intubation is done. The assistant known by different names such as "theatre assistant" "theatre technician" or "anesthetic technician" does the very same important job of assisting intubation with adequate knowledge and skill.

He must have the knowledge of:
- The name of the devices used for intubation.
- The location of the devices required.
- The basic anatomy of the upper airway with particular reference to the larynx.

He must have the skill to:
- Apply cricoid pressure to occlude the esophagus and to push the larynx posteriorly for the anesthetist to visualise it well.
- Place the required device into the hands of the anesthetist.
- Keep ready the suction and operate it when needed and apply it.
- Compress the reservoir bag properly to ventilate the patient when the anesthetist's hands are otherwise engaged.

Position the patient's head by addition or removal of pillows. Holding the head of a baby and stabilizing it when a pediatric patient is intubated.

Manipulate the larynx according to the instruction of the anesthetist.

Rarely looking down the laryngoscope blade and insert the endotracheal tube while the anesthetist is holding the laryngoscope and larynx in an optimum position.

Palpating the pulse for its regularity and rate. Check the B.P., Inflate the endotracheal tube cuff just sufficient to prevent leak.

Some of the conditions where there may be difficulty in intubation:
- A short muscular neck with full set of teeth.
- A receding mandible or a smaller mandible.
- A long high arched palate and a narrow oral cavity as in Morfan's syndrome.
  Protruding upper incisors (rabbit teeth)
- Relatively large tongue which obscures the faucial pillars, uvula and soft palate.
- When the thyro-mental distance is less than 6 cm.
- Difficulty in opening the jaw, arthritis of tempero-mandibular joint or inter dental wiring.
- Very large breasts or morbid obesity.
- Limited neck extension as in cervical spondylosis, fusion of atlanto-occipital joint, calcification of interspinous ligament, burns contractures of front of the neck and cervical spine injuries.
- Contractures in the mouth.
- Tumors of mouth and larynx.
- Acromegaly.
- In children, syndromes such as Pierre-Robin syndrome.
- Though this is not an exhaustive list of conditions causing difficulty in intubation, these have to be looked for in the preoperative evaluation.
- An easy method for any one, (need not be an anesthetist) who wants to assess whether a patient is likely to have difficulty for intubation, the following three points may be helpful. If all the three points are

clear, it can be presumed that there will not be any difficulty for intubation.

- Movements of head and neck together must be more than 90 degrees. The movement of atlanto occipital joint and movement of the neck put together.
- Thyromental distance must be at least 4 fingers. Not less than 6 cm.
- When the patient is sitting erect in front of the observer and opens the mouth as much as possible, the observer must be able to see the posterior pharyngeal wall.

Some equipments to be kept ready to meet any difficulty in intubation:

- Two laryngoscopes in working order with assorted blades. If one fails the other can be used without delay.
- Magill's intubating forceps.
- Gum elastic bougies.
- Malleable stylets of different sizes.
- Oro-pharyngeal and Naso-pharyngeal airways.
- Various sizes of endotracheal tubes. When the appropriate size for that patient does not go in, a relatively smaller may enter the glottis and save the airway.
- Suction apparatus with assorted suction tubes.
- An emergency kit for transtracheal ventilation.

A few words about extubation:

- Many of the anatomical difficulties listed above will still be present after any operation, may cause more difficulties due to depressed reflexes or drowsiness and will add to the danger.
- Hence extubation should be done only when the patient can maintain airway and breathing.
- Thorough oropharyngeal suction is done to clear the pharyngeal secretions before extubation. Endotracheal

suction need not be applied unless indicated, as this is one of the causes for atelectasis.

- There is a possibility of the patient developing Laryngospasm soon after extubation.
- Administering 100% oxygen through a mask and removing pharyngeal secretions by suction usually clears it.
- If any difficulties were encountered and managed, the difficulty and the solutions should be recorded in the anesthetic record preferably in red ink or a different colour so that it does not go unnoticed in future.

Special Care to be Taken during Intubation

- It may be unwise to abolish spontaneous respiration in patients with anticipated difficulty in intubation, before intubation without checking that manual ventilation is possible using a face mask.
- Though intubation is a technically easy procedure, it is not an innocuous technique and so it should not be undertaken casually.
- It should not be done without proper indication.
- Proper care should be taken to set all the equipments needed for intubation including availability of a good working suction with the right size suction tubes and adequate source of oxygen supply.
- The most important precaution is "Never attempt to intubate a patient alone without the availability of a well-trained, skillful theatre assistant by your side to help you" It has been well-documented that many deaths have occurred because of the nonavailability of such help.
- Intubating a patient even when no one else is available is accepted in resuscitation of an accident victim.

- The procedure of intubation cannot be taught in the form of theory material, it needs a teacher to explain every step and to demonstrate the steps. However the pictures showing the steps of the procedure are shown here to make the understanding a little more clear.

Securing and Stabilizing the Endotracheal Tube after Intubation

- The endotracheal tube after intubation has to be secured in position with the help of adhesive tapes or special harness available for this purpose.
- There is always the possibility of movement of the tube. The tube may move too much into the trachea or may move out of trachea. Both the situations are hazardous. So it is essential to fix the tube securely to prevent the movements.
- Whenever the endotracheal tube fixation is removed, two persons must be available, one to hold the tube to prevent dislodgement and the other to perform oral and nasal care.
- Inflation of the cuff is only for the purpose of making an airtight seal with in the trachea and should not be taken as a measurer of securing the tube.
- There are many methods of fixing the tube and each institution may have their own protocol for that, all aiming at stabilizing the tube in position.
- It is better to use two tapes of adequate length to fix the tube. Usually about 11 inches length for an average adult. One tape fixes the tube to the maxilla (Fixed structure) and the other fixes the tube to the mandible (unstable structure). However both the tapes together will anchor the tube in position.
- The breadth of the tape is of concern. It should the same as the diameter of the tube. For example, an 8 mm size

tube will be fixed with a tape of 8 mm width. If the width is larger than the diameter, it will form tenting near the lips and allow movement where as if it is smaller, it will cause poor fixation of the tube.
- A soft bite block may be kept in between the teeth to prevent the patient biting the tube.
- Nasal endotracheal tube is fixed to the upper lip and not the nose for anchoring.
- It is better to reposition oral tube every 24 hours to prevent pressure necrosis.
- Endotracheal tube should be fixed separately and not with any other tube, for example Ryles tube.

Intubation in Conscious Patients

A patient who is fully awake with oropharyngeal reflexes, intubation of the trachea may be done after anesthetizing oropharyngeal and laryngeal mucosa by spraying with local anesthetic (Xylocaine) 4% or 10% spray (Figs 12.28 and 12.29) after explaining the procedure to him. If properly carried out, it is well-tolerated by the adult patients.

Total dose of Xylocaine should not exceed 3 mg/kg

For nasal intubation, nasal mucosa also is to be sprayed well-preferably with xylocaine 4% mixed with 1 in 200,000 solution of adrenaline for vasoconstriction. This causes the mucosa to shrink and makes the nasal passage roomier. Incidentally chance of bleeding is less.

Through nasal route, the endotracheal tube has to be directed into the trachea using the specially designed Magill's intubating forceps (Fig. 12.30).

Fig. 12.28: Laryngeal spray (Macintosh)

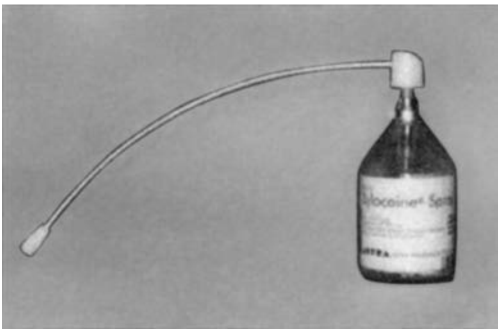

Fig. 12.29: 10% Xylocaine spray in sealed bottle with sprout and calibrated dispensing device

- ❖ This allows 10 mg of Xylocaine to be delivered each time it is pressed.

Forrester Spray (Macintosh Laryngeal Spray)

Container can hold only 4 ml of 4% xylocaine solution. This prevents accidental overdose. Restricts the dose to 160 mg.
Works on venturi principle.

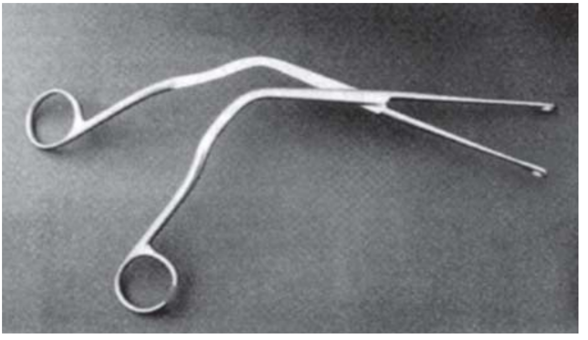

Fig. 12.30: Magill's intubating forceps. Very essential equipment for nasotracheal intubation

A Malleable sprout is used for spraying the nasal cavity, oropharynx, larynx and laryngeal inlet for topical analgesia.

EXTUBATION

- Endotracheal tube is an essential artificial airway mostly used for mechanical ventilation because it is a perfect airway available.
- It has many demerits in respiratory physiology which has been discussed well in chapter 2 and 3. So, it may be used only as long as it is essential.
- When the indications for intubation have resolved and the patient is stable, extubation may be done.
- The procedure must be explained to the patient.
- Suctioning of the tracheobronchial tree if needed is done.
- After giving enough time for the patient to settle after the suctioning, oropharyngeal suctioning is done to clear any secretions collected in between the cuff and vocal cords. If this step is not done these secretions may be aspirated into the tracheobronchial tree.

- The adhesive tape is removed, the patient is instructed to take a deep breath, and the cuff is deflated.
- Then patient is told to forcefully exhale, while the tube is removed in one swift movement. The extubation is done during expiration because the vocal cords open widely during expiration and it prevents injury.
- Patient may be asked to cough and speak after extubation to assess these functions.
- Supplemental humidified oxygen and may be necessary.
- If the patient develops signs of upper airway obstruction due to mild edema, cool mist of nebulised adrenaline may be used.
- Most important is to closely watch the patient for obstruction for many hours to say airway is normal.

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CHAPTER 13

Complications of Mechanical Ventilation

- *Early complications*
- *Delayed complications*
- *Positive pressure related problems*
- *Artificial airway related problems*
- *Ventilator associated pneumonia*
- *Oxygen toxicity*
- *Psychological and socioeconomical complications*
- *Complications attributed to operation or operator of ventilator*
- *Monitoring the patient*
- *Monitoring the ventilator*
- *Key board of a ventilator—Control panel and display panel*

PROBLEMS OF MECHANICAL VENTILATION

Though mechanical ventilation is a life saving technique, saved millions of lives which would have been lost otherwise, it is not free from problems and complications. Introduction of a machine in any clinical settings needs mastery of skills associated with its use and has its potential complications also.

It has to be remembered that it is a gross tresspass into the natural process of respiration in various forms. The first may be the bypassing of normal protection from the nose and upper airway; it continues up to the alveolar capillary membrane.

Mechanically ventilated patients are at high-risk of mortality, not only from the primary condition that necessitated ventilator support, but also from complications arising directly or indirectly from mechanical ventilation itself.

The most important change is the normal negative pressure inspiration is modified to positive pressure inspiration. This generates a series of events leading on to

may problems or complications which may be discussed in this chapter briefly.

Most of the problems may be classified into certain two main groups namely;

- Early complications
- Delayed complications

Early complications are directly related to the mechanical ventilation and problems in the airway.

1. **Early complications** are more common and are life threatening, may be due to,

- Positive pressure in the airway
  - Ventilator induced lung injuries
  - Reduction in cardiac out put
  - Alteration in renal function
  - Increase in intracranial tension
  - Ventilation perfusion mismatch.
- Artificial airways
  - Endotracheal tube obstruction.
  - Drying of airway and encrustation
  - Migration of endotracheal tube
  - Self-extubation
- Trauma to airway
- Psychological complications

2. **Delayed complications** are due to secondary effects such as infection, pressure necrosis of tracheal mucosa or oxygen toxicity, etc.

- Sinus infections
- Nasal mucosal necrosis
- Tracheoesophageal fistula
- Erosion into innominate artery
- Ventilator associated pneumonia
- Oxygen toxicity
- Psychosocial complications

POSITIVE AIRWAY PRESSURE RELATED PROBLEMS (VENTILATOR INDUCED LUNG INJURIES)

In positive pressure breathing, the intrathoracic pressures particularly, *the mean airway pressure* and *the peak airway pressure* can often rise appreciably and can have potentially harmful effects. Basically it is expected to cause three effects, Barotrauma, Volutrauma, and Biotrauma. Apart from this, it has its effect on cardiovascular system.

Barotrauma

- It is a stretch injury caused by acute rise in the airway pressure. This may be caused by excessive pressure or excessive peak inflating volume (volutrauma) or both.
- High peak inspiratory pressure (PIP) is positively correlated with air leaks, which is more common when the PIP is greater than 50 cm H_2O. Such pressures are likely when gas is unevenly distributes in the lung as in ARDS.
- When the alveolar pressure rises relative to the pressure in the interstitial space alveolar rupture may occur and air enters the pulmonary interstitium. The escaped air follows the path of least resistance and it dissects along the perivascular sheaths towards mediastinum (Pneumomediastinum). The mediastinal pleura may eventually rupture resulting in pneumothorax.
- The pneumothorax as a complication in a patient on ventilator is invariably a tension pneumothorax which is a life-threatening situation and will rapidly cause cardiovascular collapse due to mediastinal shift with kinking of all great vessels and bronchial tree. This needs immediate identification and intervention to release the tension by putting in a wide bore needle in the second intercostal space just lateral to the midclavicular line (discussed in Chapter 6).

- When the air in the mediastinum dissects up and reaches the subcutaneous tissues of neck or chest wall and cause subcutaneous emphysema.
- From the mediastinum the air may dissect the pleural reflections of great vessels and into the pericardium (pneumopericardium).
- Subcutaneous emphysema and Pneumomediastinum are not likely to cause a serious problem to the patient. However, if it is not released by proper incisions, it may progress with time.
- The air from mediastinum may dissect down wards the facial planes to reach peritoneal cavity to cause pneumoperitonium.

Prevention

- Lung damage may be minimised by avoiding overdistension of alveolar units.
- Peak and mean airway pressures may be reduced by:
  - Decreasing the tidal volume(V_T).
  - Reducing the level of PEEP.
  - Decreasing the peak inspiratory flow rate.
  - Altering the I : E ratio.
- Gas distribution may be improved by the use of a decelerating flow wave form and bronchodilators.

Volutrauma

- Large tidal volumes have a damaging effect on the lung apart from the positive pressure they produce (barotrauma). It is because of alveolar overstretching at end expiration, rather than high airway pressure.
- It is likely to causes increased epithelial and endothelial permeability and diffuse alveolar damage with

pulmonary edema, a condition indistinguishable from ARDS.

Prevention

- In ARDS, volutrauma may be reduced by using *low tidal volumes*, 6 ml/kg or lower.
- Simultaneously *applying PEEP* to keep the alveoli from collapsing prevents the need for excessively large pressure to open up the collapsed alveoli.
- PEEP, by obviating need for repeatedly opening the alveoli with each inspiration reduces the sheering forces that operate at the interface between collapsed and patent alveoli.

Biotrauma

- It is a recently recognised type of lung injury.
- Inflammatory cytokines are released from the lung in response to alveolar overdistension.
- These cytokines, which appear to be predominantly produced by alveolar macrophages, are thought to seep from the lungs to the systemic circulation. This explains how mechanical ventilation by itself contributes for organ failure by affecting other organ systems.
- It appears that the best strategy is to open up the lung (recruiting more alveolar units) and hold it open, using low tidal volumes and generous amounts of PEEP.

Reduction in Cardiac Output and Oxygen Delivery to Tissues

- Positive pressure ventilation results in hypotension because of reduced cardiac output.

- This commonly occurs when the lungs are very compliant and the chest wall is noncompliant, where more positive pressure is transmitted to the mediastinal structures preventing the venous return to the right heart.
- This hemodynamic effect is more common in patients with hypovolaemia, poor cardiac reserve and also when PEEP is applied.
- Three mechanisms play in the reduction of cardiac output. (1) Reduction of venous return to right heart. (2) Pulmonary vascular resistance is increased by the increased lung volumes resulting in right ventricular afterload. (3) Increased right ventricular afterload increases the right ventricular end systolic volume causing the bulge of interventricular septum into the left ventricle reducing its filing volume.

Management

- Increasing the cardiac output by using adequate volume of fluid to increase the preload.
- Using inotropic agents to increase the stroke volume.
- Using the modes that allow more spontaneous breathing or lower peak inspiratory pressures.

Alteration in Renal Function

- Renal blood flow may be reduced as a result of reduced cardiac output.
- Increased pressure in renal vein may shift the flow from cortex to medulla enhancing sodium reabsorption and decreased filtration and so the urine output.
- Because of fall in renal blood flow, the renin-angiotensin-aldosterone system is stimulated to retain additional sodium and water.

- ADH secretion from posterior pituitary is stimulated by vagal receptors in the right atrium that senses the decrease in venous return as hypovolaemia. ADH promotes fluid retention and reduced urine out put.
- Atrial natriuretic peptide (ANP) is decreased. This is a natural diuretic that inhibits the secretion of aldosterone and renin. So, less circulating natural diuretic and more aldosterone results in sodium and water retention.

Management

- When ever necessary, diuretics may be used to reduce water retention.
- Patient's weight must be frequently monitored to allow fluid balance.

Increase in Intracranial Tension

- Positive pressure respiration has no adverse effects on intracranial pressure (ICP) in individual with normal cerebrovascular hemodynamics.
- However, in patients with altered cerebrovascular hemodynamics due to injury or intracranial pathology, when positive pressure ventilation (IPPV) is instituted, particularly with PEEP, there will be increase in intracranial pressure (ICP) and decrease in cerebral perfusion pressure (CPP) occurs. The pressures in superior vena cava and jugular vein increase and diminish the cerebral venous out flow that results in increase in ICP.
- The cerebral perfusion pressure (CPP) may further be decreased when there is a decreased cardiac output (CO) secondary to the increase in intrathoracic pressure in IPPV.

Management

- Infusing large volume of preloading in an attempt to restore normal hemodynamic status must be avoided.
- Overall high mean intrathoracic pressure should be avoided especially in patients with altered cerebrovascular status.

Ventilation Perfusion Mismatch

- Mechanical ventilation strategy should optimse the V/Q. In nonuniform disease processes in which the V_T is maldisrtibuted, alveoli are venerable for overdistension.
- Alveolar overdistension caused by elevated mean airway pressure, large tidal volume and use of PEEP.
- This may cause compression of adjacent pulmonary capillaries and regional hyper perfusion thus results in ventilation/perfusion (V/Q) mismatch.

Management

- Use of low tidal volume (V_T) with higher respiratory rate (RR) and reduction in PEEP are well-tolerated.
- Either decelerating or accelerating flow wave-pattern may be used.

ARTIFICIAL AIRWAY RELATED PROBLEMS

- For maintaining mechanical ventilation, securing the airway of the patient with an artificial airway is essential. Most commonly *endotracheal tube* is used either through nasal or oral route.
- *Endotracheal tube narrowing* can occur commonly due to encrustations which occur at the lumen of the tube near

the tip. The mucociliary escalator brings the mucous towards the larynx and it stops at the level of the tip of the tube and the mucous collects around the tip of the tube. With each respiratory cycle the secretions move in and out of the tip of the tube, getting dried up slowly forming encrustation. These encrustations at the tip of endotracheal tube forms circumferential narrowing of the tip of the tube.

- Some believe that it is a delayed problem, but this occurs in a surprising rapidity with in a day or two after instituting ventilator support especially when the lung has retained secretions with infection.
- A suction catheter passed down the tube gets impeded by this blockade. If the endotracheal tube cannot be unblocked immediately, it must be removed as an emergency and replaced with a new one with all precautions.
- *Increased resistance* to respiration caused by the narrowing of the tube eventually increases the peak inflation pressure.
- *Endotracheal tube obstruction* can be caused acutely if a small spec of mucous is lodged in this narrow lumen.
- *Drying of the airway* that leads to encrustations occurs in spite of all efforts to maintain the humidification of inspired gases at the best level. This may increase the resistance to gas flow.
- *Endotracheal tube migration* can occur either upwards or downwards. Normally the tip of endotracheal tube should be placed 2 cm from the carina. Flexing the neck migrates the endotracheal tube downwards whereas in extension of the neck has the opposite effect.
- Improperly secured endotracheal tube is likely to migrate either too much into the trachea or move out of trachea. Moving in might result in right sided

intubation as the right main bronchus is more aligned with trachea. Similarly, any drag on the tube may cause that to move out, sometimes resulting in accidental extubation.

- *Self extubation* is not an uncommon complication. Removal of endotracheal tube in a mechanically ventilated patient may prove catastrophic. This happens more frequently in patients with disorientation who are uncooperative. This dangerous complication can be prevented by adequate sedation and making the patient comfortable by using appropriate ventilator settings to the patient's needs.
- *Cuff leak* is another frequent complication that can occur in any patient on ventilator. The major problem associated with is, allowing the secretions collected above the cuff to trickle down into the tracheobronchial tree, increasing the chances for ventilator associated pneumonia. There can be sudden loss of airway pressures particularly when PEEP is used.

Prevention

- All the problems and complications can be prevented by giving a protocol based care of the endotracheal tube and checking for the specific problems that are common in that particular clinical situation.

DELAYED COMPLICATIONS

Sinus Infection (Sinusitis)

- Collection of nasal secretions due to depressed natural process of ciliary activity, posture, presence of an endotracheal tube, a nasogastric tube, etc. predispose to the sinusitis.

- This complication occurs both with nasal or oral intubation, however, it is common with nasal intubation.
- Bacterial sinusitis is one of the important causes for nosocomial pneumonia.

Nasal Mucosal Necrosis

- Presence of an endotracheal tube in nasal cavity for prolonged period is frequently associated with necrosis of the mucosa.

Tracheoesophageal Fistula

- Though it is said to occur in 1% of patients, it is a relatively uncommon and associated with high mortality.
- High pressures in the cuff of endotracheal tube compromise the capillary perfusion in the tracheal mucosa that leads to ischemic necrosis. The mucosal injury is quite insidious and a fistula is not recognised until it is quite big.
- Unusual leak of air and substantial increase in the quantity of secretion arouses doubt of a fistula.

Prevention

Since tracheoesophageal fistula produced considerable morbidity and the treatment is equally difficult, all care must be taken to prevent its occurrence.

- First step is using high volume, low pressure cuffs, preferably a square cuff rather than fusiform cuff. These cuffs produce minimal pressure that does not occlude capillary perfusion to tracheal mucosa at the same time give air tight fit on larger area of mucosa.

- Second strep is to carefully monitoring the cuff pressure by using cuff pressure gauges.
- For long time ventilation, it is advised to deflate the cuff periodically after carefully clearing oropharyngeal secretions (toileting).Deflating for a period of 10 minutes will be adequate for release of pressure on the mucosa. This procedure has its own risks.

Ventilator Associated Pneumonia

The basis of this potential complication can be is discussed briefly here.

- The principal mechanism for the development of nosocomial pneumonia appears to be aspiration of gastric and oropharyngeal organisms, primarily colonised gram- negative bacteria into the tracheobronchial tree.
- Placement of an endotracheal tube forms a conduit for contamination of the lower airway.
- As discussed earlier in anatomy, 300 million alveoli with a surface area of about 70 square meters of alveolar capillary membrane are open to the atmosphere and is vulnerable for contamination with repairable size particles.
- Particles from 0.5 microns to 2 microns can reach and be deposited in the terminal bronchioles. The bacteria are of this size, but normally very few bacteria reach this area because of the natural protections available.
- However, most bacteria causing pneumonia first colonise in the upper airway and then descend to the lower respiratory tract.
- The upper airways are virtually teaming with micro-organisms and most of them flourish even in good health. Majority of them are anerobes and they outnumber the aerobes by about three to five times.

- In healthy adults, upper respiratory secretions generally contain 10 million to 100 million organisms per ml of secretions.
- During sleep in 45% of healthy adults, small quantities of upper airway secretions are aspirated into tracheobronchial tree.
- Much larger percentage of adults aspirate pharyngeal secretions during sickness. In sickness, not only the aspirations are more frequent, but the flora is also different.
- The incidence of colonization of gram-negative bacteria rises with the gravity of illness as well as with the degree of supportive care required.
- Once the gram-negative aerobes colonise in the oropharynx, there is more chance for aspiration of these noxious organisms into the lower respiratory tract, with potential risk of nosocomial pneumonia.

Prevention

- Factors contributing to infection that need to be taken care of are:
  - Poor oral hygiene
  - Contaminated respiratory therapy equipments
  - Poor hand washing by the care givers
  - Breach of aseptic techniques during suctioning
  - Trauma during suctioning
  - Impairment of the mucociliary system because of oxygen toxicity
  - Inadequate hydration
  - Suboptimal humidification
  - Poor nutrition
- The condensate in the circuit tubing contains loads of organisms.

- This must be considered as an infectious waste and handled properly. *It should not be drained into the humidifier* because it will contaminate the entire water reservoir.
- Respiratory therapy equipments must be adequately cleaned and decontaminated. Equipment changes must be made as per scheduled protocol.
- Suctioning technique must be proper, as it is a procedure that can easily transmit infection into the respiratory tract. Proper hand washing must be done and care must be taken to prevent aspiration.
- Finally the adequate nutrition to protect and maintain the host defence.

Oxygen toxicity

This has been discussed in the Chapter 5.

Psychological and Socioeconomical Complications

- There is always a fear among public that when a patient is connected to a ventilator, there is no hope for recovery. It must be made clear that it is a treatment modality and like any treatment, this also is required for a duration needed according to the type of underlying disease. Very rarely, the underlying disease may not get reversed or may get worsened. In such circumstances it may not be possible to wean the patient from the ventilator. The physician must clearly discuss these aspects to the close kin, not only for making them to understand the facts, but to prevent future litigations based on lack of information.
- First and fore most is the anxiety in the patient and his family related to the disease process, the investigations,

prognosis, mechanical ventilation, etc. This is not only related to psychological aspect, but the economical aspect also, mostly related to the financial burden to the family.

- Many patients develop "claustrophobia" because of their inability to communicate to others.
- Many patients lose the orientation of day, night, date, time, etc.
- Imposed immobility may cause physical discomfort and frustration.
- Many individuals view "artificial ventilation" as a grave thing and believe that the patient may not survive.
- Many times the alarms are also responsible for frightening them.

Management

- The clinician must provide frequent repetitive information which must sound in such a way to create a feeling of belief in the patient.
- Physician must communicate verbally with the patient in a very clear way that he understands it and looking at his facial expressions, physician may put leading questions and give the answers.
- Periods of uninterrupted sleep must be supported and meaningless, unnecessary talk and noise avoided.
- A clock and a calendar may be placed n the view of the patient for his orientation of time and date.
- Some means of communication such as a slate and pencil, a paper and a pencil must be provided for the patient to write what he feels. Reassure the patient very firmly that the loss of voice is temporary and he can talk once the tube is removed.

Complications Attributed to Operation or Operator of Ventilator

Though this aspect is not discussed commonly, this one among the main reasons for complications and failure of therapy. This may result either because of carelessness or lack of knowledge of ventilator functioning.

- Because, it is likely that human errors or machine errors may result in failure to ventilate the patient, as an emergency measure, a manual resuscitation bag with oxygen flow meter must be available at the bedside of every patient ventilated mechanically. This can be used in the event of any problem.
- Inaccurate settings, incompatible settings, incorrect assembly, and failure to set and activate alarms.
- The patient and the ventilator settings must be checked at least every 4 hours.
- Electrical and pneumatic failure may result in failure of ventilating the patient.
- Accidental disconnection of ventilator is a common problem. The site of disconnection is usually the machine patient interface- the endotracheal connector. This is more so with patients with tracheostomy tube, as the connections are made gently to avoid discomfort to the patient.
- The next common site of disconnection is at humidifier.
- The ventilator circuit must be carefully secured in suitable clamps to prevent them dragging on the tube and connector.

Prevention

The complications can be prevented by continuous monitoring and frequent assessment of the patient and ventilator by a systematic approach.

- Evaluate the patient's response to the current level of support.
- Decide about the accuracy and appropriateness of the current ventilator settings.
- Making sure that necessary equipments are available.

Monitoring the Patient

- Patient should be assessed every two hours.
- Traditional examination of inspection, palpation, percussion, and auscultation must be done.
- Observation of the chest expansion to check whether it is equal in both sides can be done by watching the chest from the foot end of the bed.
- *Vital signs:* blood pressure, heart rate and rhythm, temperature and respiration; including patient and ventilator rates, pattern and depth.
- *Volume status*: pulmonary artery catheter reading, urine out put, intake output. Finally monitor the effects of respiratory cycle on the interpretation of hemodynamic parameters.

Laboratory values:

- Monitor arterial blood gas (ABG): for oxygenation, ventilation, and acid-base status.
- Estimate serum potassium, sodium, magnesium, and phosphorus concentrations.
- Determine end tidal CO_2
- Assess the level of consciousness
- Mental status of the patient, anxiety, fear, restlessness, agitation, confusion.

MONITOR THE VENTILATOR

Key Board of a Ventilator

- The key board of a ventilator is usually divided into three areas namely, *display panel* for patient data, *control*

panel for ventilator settings, and *status panel* for alarm conditions. Different manufacturers may have slight modifications in that.

- **The control panel** is where; the ventilator settings and alarm parameters are established.
- **The display panel** provides patient information such as exhaled tidal volume (V_T), minute ventilation (V_E), peak inspiratory pressure (PIP), etc.
- Ventilator should be checked systematically every 4 hours in a scheduled protocol.
- ABG values must be checked after 20 minutes of every change in the ventilator setting, to allow time for the parameters to change.
- Settings:

Monitor the information on the **control panel**.

- F_IO_2: Inspired oxygen concentration
- Set rate: Respiratory rate
- V_T : Tidal volume
- Level of PEEP: Positive end expiratory pressure
- Level of pressure support or pressure control: PS or PC
- Peak inspiratory flow rate and wave form:
- I:E ratio or percentage of inspiratory and expiratory times:
- Sensitivity: Trigger setting
- *Patient data:* Monitor the following information on the **display panel**.
  - Peak, Mean and Plateau airway pressures.
  - PEEP:
  - RR: Respiratory rate of ventilator and patient.
  - Exhaled tidal volume (V_T): Mandatory and Spontaneous breaths.
  - Minute ventilation (V_E):
  - Measure compliance, resistance, vital capacity.
  - Measure total PEEP to assess whether there is auto PEEP.

Alarms

- Ensure that all alarms are activated and appropriate alarm limits are set.
- They may provide audio or visual or both warnings, but should never be taken as fail safe.
- When any ventilator alarm sounds, the first thing to do is to look at the patient.
- If the patient is disconnected from the machine, reconnect him to the ventilator.
- If the patient is connected to the ventilator, but still he is in distress, the cause could not be identified immediately then, disconnect him from ventilator and manually ventilate him. Mean while call for help to identify the problem.
- Finally, if the alarm sounds and the patient is not in distress, determine which alarm sounds and proceed with problem solving.
- For problem solving, one has to look into the control panel and display panel of the ventilator.

PATIENT-VENTILATOR ASYNCHRONY

- This is commonly termed as patient “Fighting the ventilator” or patient is “bucking” the ventilator.
- It indicates that the patient has acute respiratory distress and the patient and ventilator are breathing out of synchrony with one another.
- It may be manifested with many signs such as distress, agitation, use of accessory muscles, tachypnea, abdominal paradox, hypertension, tachycardia, sweating, etc.
- Multiple alarms may sound, high pressure limit, low tidal volume, etc.
- The primary goal is to provide adequate ventilation and oxygenation. Therefore, the patient is disconnected

from the ventilator and ventilated manually with 100% oxygen.

- The causes may be either patient related or ventilator and related.
- Patient related causes:
  - Migration of tube to one side.
  - Increased resistance; Bronchospasm or secretions.
  - Decreased compliance; Tension pneumothorax or pulmonary edema
  - Development of auto PEEP
  - Pulmonary embolus.
- Ventilator related causes:
  - Sensitivity is too high or too low.
  - Inadequate peak inspiratory flow rate setting.
  - Inadequate ventilatory support
  - Inadequate supply of oxygen.
  - Large leak of air in circuitry.

Careful identification of cause and treating the causes is the corner stone of management of this problem.

PULSES OXIMETER (Fig. 13.1)

- It is the continuous estimation of the percentage saturation of hemoglobin with oxygen (SaO_2) by a noninvasive method. The normal value is between 95 to 97 %. This is the amount of oxygen carried in the blood.
- All patients on ventilator must have pulse oximeter attached.
- This is one of the most important and versatile inventions in electronics in the field of noninvasive monitoring that had saved many lives in the past two decades. It was invented and introduced in clinical practice in 1983.
- This equipment *uses pulsatile blood flow in the arterioles* for calculating the oxygen saturation it counts the pulse rate also.

- Hence any basic models of pulse oximeter will show *oxygen saturation* and *pulse rate* on display.

Display in an ideal pulse oximeter may be:
- The display is LCD (Liquid crystal display) and not LED (light emitting diodes).
- The display must be on a backlit screen of soothing colour like blue of green.
- The main display figures of saturation and pulse rate must have a size of at least 1 cm height, so that it is clearly visible at a distance of 10 feet in a relatively low intensity of ambient light.
- The alarm setting for both oxygen saturation as well as pulse rate must be available on the display.
- When an alarm sounds, the display of the particular parameter for which alarm is triggered must blink.
- In sophisticated machines there will be display in words which also blinks (e.g., Pulse High).
- Apart from this, there will be *a visual alarm of a red light blinking*, to attract the attention of the physician in case the audible alarm is of low volume and is not noted.
- Ideally, some sophisticated machines have a "perfusion indicator" or "Signal Bar" which give an indication about the degree of perfusion to the peripheral part to which the probe is attached.
- The presence of a pulse waveform is essential. This is known as pulse plethysmography. No pulse oximeter should be used unless a plethysmograph trace is displayed.

PULSE PLETHYSMOGRAPHY

- Some monitors have a plethysmographic representation of the pulse in a wave-form which will give an idea about the arterial pulse and its waves.

- Pulse Plethysmography is based upon the measurement of the increase in the volume of an extremity, usually a finger or an ear lobe, during or shortly after systole.
- All Pulse Plethysmograph devices now use the technique of photo-plethysmography. A low level of electromagnetic energy (infra red light) is passed through the extremity.
- Most of the energy, which must be at a wavelength to which the part is translucent, is detected by a semiconductor sensor or photodetector.
- An increase in volume of the part is then detected as an increase in absorption of the incident light during systole.
- The signal from the photodetector is then amplified and may be displayed on the screen. This technique is so sensitive that the dicrotic notch is easily visible.
- If the pulsatility of the signals decreases below a critical level, the alarm is initiated.

Fig. 13.1: A pulse oximeter with all the essential features on the screen

It works on the following principle:

- Absorption spectra of oxyhemoglobin and that of reduced hemoglobin are different and by calculating the values of each in the arterial blood (arteriole) the percentage of O_2 saturation can be deducted.
- The Beer-Lambert law forms the basis of spectrophotometric techniques such as oximetry.
- It is the combination of the two laws that describe the absorption of monochromatic light by a transparent substance through which it passes.
- *Beer's law:* intensity of transmitted light decreases exponentially as the concentration of the substance increases.
- *Lambert's law:* intensity of transmitted light decreases exponentially as the distance traveled through the substance increases.
- During a pulsatile flow in the systolic phase there will be a slight increase in oxyhemoglobin levels than in the diastolic phase.
- This small difference is sensed by the sensors and processed. So the venous blood is ignored.
- Therefore, it cannot sense the saturation in nonpulsatile flow as in extracorporeal circulation.
- There are two light emitting diodes (LED) in the probe which emit light with wave-lengths of 660 nm (Red) and 940 nm (Infrared).
- The two LEDs alternatively emit light which passes through the tissues in the finger tip.
- The light which comes out on the other side of the finger after being absorbed by various tissues is picked up by a photosensor called the photo diode.
- The light passing through the pulsatile structures (arterioles) have a minimal difference between the systolic and diastolic phase which will be picked up

by the sensor and sent to the microprocessor for processing.

- The nonpulsatile structures like tissues, bones, tendons, and nails also absorb light but as there are no differences they are ignored by the sensor and not processed.
- Therefore, depending on the wavelengths of the light absorbed by the oxyhemoglobin and reduced hemoglobin, the microprocessor calculates the percentage saturation and displays it digitally on the screen.
- The red light is readily absorbed by deoxygenated (reduced) hemoglobin and the infrared light by the oxyhemoglobin (HbO_2).
- The pulses oximeter measures the deoxygenated hemoglobin (Hb) and oxygenated hemoglobin (HbO_2). Total hemoglobin is the sum of Hb and HbO_2.
- Hence, the SpO_2 value is the percentage of total hemoglobin which can be oxygenated.
- Some types of hemoglobins like carboxyhemoglobin are bright in color and have an absorption wavelength closer to oxyhemoglobin . This may be interpreted as oxyhemoglobin.
- Similarly, some types like methemoglobin are darker in color and hence may be interpreted as reduced hemoglobin.
- If these are not differentiated, the % saturation value may not be real.
- This is called fractional saturation (SaO_2).
- If these fractions are detected and deleted, actual oxyhemoglobin saturation is calculated.
- The percentage of the total hemoglobin that can be oxygenated is SpO_2.
- It is called functional saturation (SpO_2).

- The formula for calculating saturation is $SpO_2 = HbO_2 / Hb + HbO_2$
- The heart rate is also displayed as the number of pulses is counted.
- The response time varies from 5-20 seconds and it can be adjusted in some models.
- Any value less than 70% carries little meaning as the calibration is done with healthy volunteers and it is impossible to bring any individual below this level safely.
- One molecule of hemoglobin carries 4 molecules of O_2.
- The absorption spectrums of adult HbA and HbF are almost the same.
- So, in a neonate, where the % of HbF is considerably higher, the adult probe can be used.

Reliability

- Pulse oximeter readings are reliable with 2% error.
- The presence in large amount of dysfunctional hemoglobin like carboxyhemoglobin and methemoglobin may give errors.
- Bright ambient light, movements of the patient, shivering, will modify the value.
- Dyes like methylene blue, indigo carmine, indocynine green may cause changes in value.
- Deeply pigmented skin may show a reduced value.
- Nail polish except black, blue and green, normally does not modify the value.
- Hence, the SpO_2 value is the percentage of total hemoglobin which can be oxygenated.
- Some types of hemoglobin s like carboxyhemoglobin are bright in color and have an absorption wavelength closer to oxyhemoglobin. This may be interpreted as oxyhemoglobin.

- Similarly, some types like methemoglobin are darker in color and hence may be interpreted as reduced hemoglobin .
- If these fractions are detected and deleted, actual oxyhemoglobin saturation is calculated.
- Low perfusion to the area such as hypothermia, hypotension, vasopressors, and application of B.P cuff may reduce the value.

Capnography: End Tidal CO_2 Value

- The amount of CO_2 in exhaled gases is determined by *metabolic rate, perfusion status* (to remove the gas from the tissues and bring it to the lungs) and *alveolar ventilation.*
- Therefore, the measurement of concentration of CO_2 at the end of tidal breath provides a reflection of the alveolar CO_2 (PAO_2), which in turn reflects the arterial CO_2 ($PaCO_2$). So, $etCO_2$ - $PACO_2$ - $PaCO_2$.
- At the beginning of expiration, the gas from the anatomical dead space that is free from CO_2 is exhaled.
- CO_2 elimination rapidly rises, reaching a plateau as the alveolar gases are exhaled.
- Therefore, the concentration of CO_2 in the expired gases is maximum or at the peak at the end of expiration (end tidal).
- It can be measured near the proximal end of endotracheal tube.
- The measurement of CO_2 concentration at the end of expiration is called Capnography.
- Noninvasive measurement of CO_2 concentration and giving in numerical display is called "Capnometry".
- The numerical and graphic display of CO_2 in respired gases in a waveform is known as "Capnography" and the display pattern on a paper is called as "Capnogram".

- The main purpose of Capnography is to monitor the patient's ventilatory status ($PaCO_2$) as the maintenance of normal $PaCO_2$ depends upon adequacy of alveolar ventilation.
- There are two methods of analyzing CO_2 in the expired gases; one is "Mass spectrometry" and the other is "Infra-red absorption spectrometry".

Mass Spectrometry

- It is used for monitoring many patients simultaneously.
- It involves sampling from the patient and transporting to the equipment through special tubing.
- Therefore, it does not continuously monitor the end tidal CO_2.
- Moreover, it is sophisticated equipment that it can monitor the concentrations of other gases like O_2, Nitrogen and even the concentrations of anesthetic agents in the gas mixture.
- It is highly expensive and is not suited for monitoring individual patient. Infrared absorption spectrometry or Infrared CO_2 analyser.
- This equipment is less expensive, portable, and most commonly used.
- It works on the principle of infrared light absorption by CO_2 in the mixture of gases.
- An IR analyser simply has a source of IR radiation, an analysis chamber, a reference chamber, and a detection cell.
- The light absorption by CO_2 in the analyzing chamber is compared to that reference chamber which has no CO_2.
- It provides continuous monitoring of the patient.

- There are two systems of sampling available based on which there are two types of monitors available; one is *mainstream analyser* and the other is *side stream analyser*.

Mainstream Analyser

- This no loss system offers a very fast response.
- It incorporates; the analyser cell with IR source, detector, and the associated electronics into a specially designed airway adopter which is interposed into the breathing system.
- The possibility of condensation of water vapor is prevented by heating the measuring chamber to about 40°C.
- The possibility of contamination with secretions that absorbs IR light may give an erroneously high value of CO_2.
- The added apparatus dead space is disadvantageous in children.
- There can be a drag on the endotracheal tube, because of the weight.

Side Stream Analyser

- The sampling is done by a small bore tube (usually made of Teflon as PVC may react with halogenated hydrocarbons) of about 2 meters length to the measuring chamber.
- The end of sampling tube is kept at the proximal end of endotracheal tube using a T-piece assembly. The sampling rate is in the order of 50 to 500 ml/min.
- The advantage is that there is no addition of apparatus dead space.

- The disadvantages include delay in the response, as the gas has to travel through the small tube to the analyser chamber and the volume loss by sampling, water condensation in the sampling tube.

Interpretation of Values

- The normal value is usually 2 to 5 mm Hg lesser than the $PaCO_2$. For example, if the $PaCO_2$ is 39 mm Hg and the $etCO_2$ is 35 mm Hg, it is normal.
  Factors that modify the $etCO_2$ value: it may be raised or lowered.

Factors that Increase $etCO_2$

- *Ventilation:* Hypoventilation due to any reason.
- *Perfusion:* Increased transport of CO_2 to lungs as in post cardiac arrest state.
- *Metabolism:* increased CO_2 production due to any reason, most commonly pyrexia, trauma, shivering, or malignant hyperthermia, etc.
- *Equipment:* Rebreathing due to any reason, partially obstructed airway, or leak in the breathing circuit delivering low volume, etc.

Factors that Decreases at CO_2

- Hyperventilation.
- Reduced pulmonary perfusion as in decreased cardiac output, pulmonary embolus, etc.
- Decreased CO_2 production as in hypothermia, heavy sedation, etc.
- Ventilator disconnection.

Normal Capnogram and Various Abnormal Patterns

- Capnogram is not a measurement of respiratory function only. It has to be interpreted in conjunction with other clinical findings. Like ECG, it requires systematic analysis with regard to the baseline, height, frequency, rhythm, and shape to get the best information.
- The striking difference of capnogram wave from other respiratory waveforms is that the positive deflections indicate expiration and the negative deflection indicates inspiration (Fig. 13.2).
- The shape of the capnogram is diagnostic of abnormal lung function or suggestive of technical problem.
- As at the beginning of expiration, the air from the anatomical dead space escapes, the CO_2 value is zero. This is baseline (A-B).
- Then there is a sharp rise of the wave when CO_2 elimination occurs as the alveolar air mixes with dead space air (B-C).
- After this sharp rise, the wave becomes a plateau. This plateau is called as "Alveolar plateau" where most of the gas flow from the alveoli occurs. The plateau gradually ascends and reaches a peak (C-D).
- This point at the end of plateau is the end of expiration and the PCO_2 at this point is the end tidal CO_2.
- Therefore, the $etCO_2$ is the highest concentration of CO_2 exhaled.
- The curve then takes a sharp down stroke as the fresh gas free from CO_2 is inhaled.

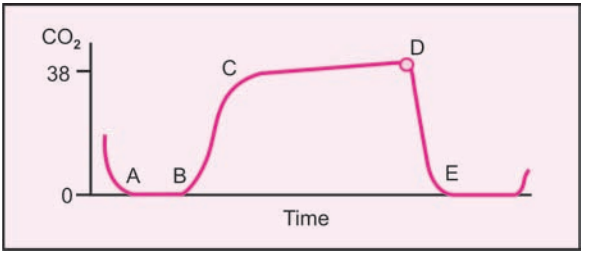

Fig. 13.2: Normal capnogram

❖	Inspiratory baseline:	**A-B**	Just before expiration
❖	Expiratory upstroke:	**B-C**	Active expiration
❖	Expiratory plateau:	**C-D**	Expiration of alveolar air
❖	Inspiratory down stroke:	**D-E**	Inspiration

Individual capnogram wave can be analysed for studying each characteristic.

A trend of capnogram over a period of time can be analysed to know the changes that has occurred during the period (Fig. 13.3).

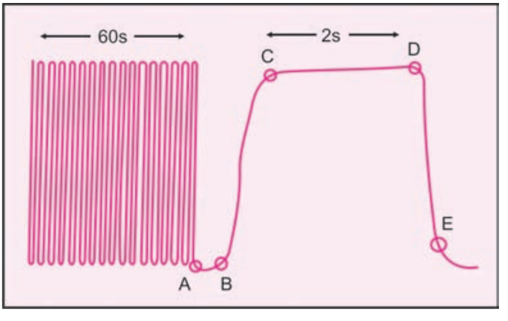

Fig. 13.3: Normal capnogram, the trend for 60 seconds and a single waveform

Some abnormal capnogram patterns and the possible causes are discussed below:

- Elevated etCO_2 with good alveolar plateau (Fig. 13.4)

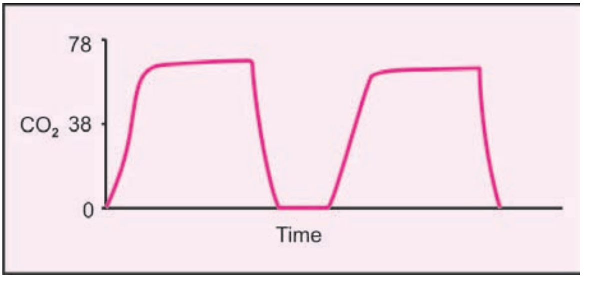

Fig. 13.4: Elevated end tidal CO_2 with a good alveolar plateau

❖ Inadequate minute volume (V_E) with tidal volume sufficient to empty the alveolar gas.
❖ Increased metabolic rate such as fever, pain, shivering.

- Progressively increasing etCO_2 with normal alveolar plateau (Fig. 13.5).

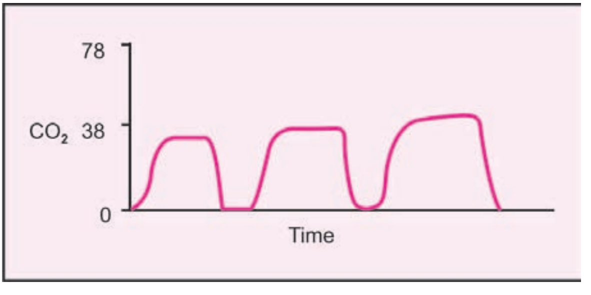

Fig. 13.5: Gradually increasing end tidal CO_2 with normal alveolar plateau

❖ Hypoventilation.
❖ Malignant hyperthermia.
❖ Factors that raise the body temperature.

- A rise in baseline and the $etCO_2$ (Fig. 13.6).

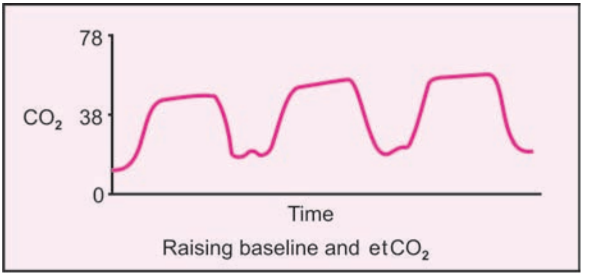

Fig. 13.6: Increase in end tidal CO_2 and elevated inspiratory baseline

❖ Rebreathing of exhaled CO_2.
❖ Increased apparatus dead space.
❖ Exhausted soda lime in anesthesia.
❖ Defective expiratory valve.

- Alveolar plateau showing an cleft on the top (alveolar cleft) (Fig. 13.7).

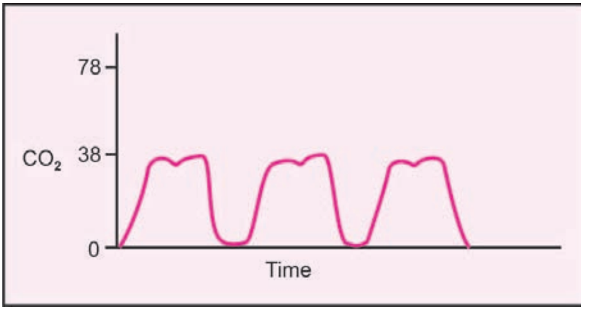

Fig. 13.7: Normal capnogram with "alveolar cleft"

❖ Inadequate neuromuscular blockade or neuromuscular block wearing off.
❖ The patient's inspiratory attempts are seen as a depression on the top of the alveolar plateau, known as "Alveolar cleft".

- Oblique expiratory upstroke (Fig. 13.8).

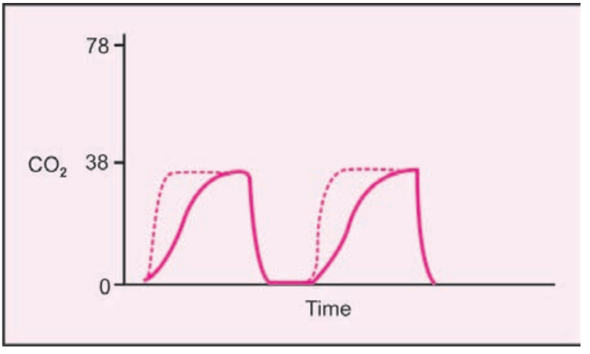

Fig. 13.8: Sloping alveolar plateau

❖ Expiratory obstruction as in bronchospasm.
❖ The airway obstruction causes a delay in alveolar air and CO_2 to be expelled, so, the upstroke is not sharp and merges with the plateau. The dotted line indicates the normal upstroke and plateau.

- Progressively decreasing end tidal CO_2 (Fig. 13.9).

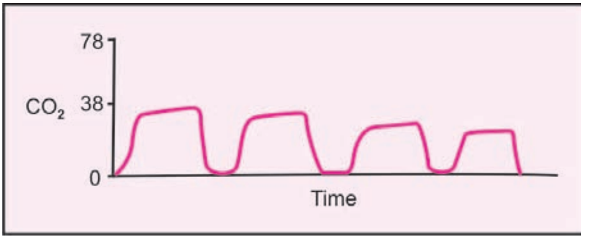

Fig. 13.9: Progressively decreasing end tidal CO_2

❖ Reduction in blood flow to the lung.
❖ Severe hypotension or pulmonary embolus.

SERUM LACTATE CONCENTRATION

Achieving maximal oxygen delivery (CO_2) in critically ill patients results in improved survival. Timely resuscitation

of oxygen deficiency states has shown to reduce morbidity and mortality in them.

Underlying oxygen deficits must be treated with the use of physiologic principles to ensure that adequate resuscitation has been done at cellular level, where the ultimate respiratory units lie.

Decreased oxygen delivery (DOi) in critically ill patients may cause inadequate tissue perfusion which may be due to hypovolemia, cardiogenic or distributive shock. This initiates anerobic metabolism and the release of lactic acid as metabolite causing lactic acidosis. Hence estimation of serum lactate concentration at regular intervals will give a orientation about the tissues perfusion at large.

The normal value of serum lactate is **0. 5 to 2.0 mEq/L.**

- Very commonly, blood levels of lactate increases with anerobic metabolism and represents the total O_2 debt or the magnitude of hypoperfusion.
- It may be increased in other conditions also. So, lactic acidosis is classified into two types.
- **Type A:** With clinical evidence of tissue hypoxia.
  - This is due to an imbalance in oxygen supply and demand.
- **Type B:** Without clinical evidence of tissue hypoxia.
  - This may occur in liver diseases, malignancy, diabetes mellitus, toxins like ethanol, and drugs like salicylate.
- Lactate is primarily metabolized by liver. The normal amount of lactate produced under aerobic conditions is generally managed by the liver. Therefore, under normal conditions of aerobic metabolism and liver functions, there is no significant accumulation of lactate in blood.

- However, if the amount of lactate produced exceeds the capability of liver to metabolise it, it may accumulate to cause metabolic acidosis.
- The patient's baseline liver function also affects the lactate clearance and must be taken into account in interpreting the serum lactate value.
- Patient's baseline lactate value is taken.
- Patient's cardiopulmonary profile is obtained before assessing the value.
- The amount of serum lactate gives an indication of the severity of O_2 debt.
- Serial lactate level estimations will be useful in assessing the effectiveness of the resuscitative therapies.
- Ideally a steady decrease in the lactate level should be seen.
- If it does not work, then systematic re-evaluation of therapy must be done to find further ways to improve oxygen delivery (DO_2) and decrease oxygen demand.
- The estimation of pH and base deficit may also help to assess the adequacy of tissue perfusion.

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CHAPTER 14

Weaning from Ventilator

- *Physiology of respiratory muscles*
- *Means to increase the strength and endurance*
- *Identifying muscle fatigue, prevention, and management*
- *Weaning criteria: Ventilation, oxygenation, mechanics*
- *Weaning modes*
- *Psychological aspect of weaning*
- *Simple weaning criteria for ICU patients (A-E)*
- *Parameters commonly used to predict successful weaning*

Weaning is a term literally related to the breastfeeding and it refers to the slow processes of changing the baby from breast milk to other feeding and ultimately stopping breastfeed. It is almost a natural process and is easy. So also, weaning from mechanical ventilation is the process of withdrawing the patient from mechanical ventilatory support in a staged manner. Nevertheless, it is not that easy as weaning from breast milk.

Weaning is really the transfer of demands from the ventilator to the patient.

"It is easy to put a patient on a ventilator support, but it is difficult to wean,"

The disease process which necessitated the ventilator support must be reversed or must be under control before attempting weaning.

Among the patients who are to be weaned from a ventilator, there are two groups.

- Those who belong to the first group were on short-term ventilator support with out any underlying disease of the lungs and respiratory muscles. In these patients, ventilatory support could be simply discontinued as they are awake and have adequate muscle strength to breath on their own. For example, elective postoperative ventilatory support.

- Those in the second group are patients in whom the respiratory muscles were weak due to disease and had inadequate muscle strength for the work of breathing. Moreover, during prolonged ventilatory therapy the muscles would have gone for disuse atrophy. (Weakened and detrained).

In these patients weaning is relatively more difficult and repeated unsuccessful attempts may be encountered. Usually the ease of weaning is inversely proportional to the duration of ventilator therapy.

The difficulty may be greater, if associated problems like malnutrition, stress, etc.

In mechanical ventilation for longer periods, respiratory muscle atrophy inevitably occurs and the muscles lose its strength and endurance. The weaning program consists of exercises to gradually recondition the respiratory muscles to take over the loads of respiratory work. Training is achieved by applying sufficient load in terms of intensity, duration, and frequency to the respiratory muscles. This will be evidenced by the ability to sustain spontaneous ventilation for increasingly longer periods.

The primary goal is to facilitate the **strength** and **endurance** of respiratory muscles for sustaining spontaneous breathing.

Strength is the ability to develop force against resistance in a single contraction.

Endurance is the ability to exercise for prolonged periods before fatigue occurs.

In other words, it is a gradual process, where the patient is given increasing responsibility for his breathing; ultimately he is allowed to do unassisted spontaneous breathing. This can be achieved by improving nutrition and application of a planned, well monitored program of

training and permitting the respiratory muscles to take over the work of breathing.

PHYSIOLOGY OF RESPIRATORY MUSCLES

Physiological aspects of respiratory muscles have to be taken into consideration for the weaning process, as they have to take over the work of breathing.

Based on their duration of contraction, the skeletal muscles are classified into two types; **Type I (**Slow twitch fibers) and **Type II** (Fast twitch fibers). Respiratory muscles are mixed muscles, for instance, the diaphragm has about 50% of Type I fibers and the remaining fibers Type II.

Skeletal Muscle Types

Type I: Slow Twitch Fibers

- These are the fibers, which has **more endurance.**
- These fibers are adapted for prolonged performance of work and are resistant to fatigue.
- They are smaller and generate lower level of force when activated.
- They have more capillary supply and have more mitochondria than fast fibers, indicating their metabolic stability.
- For any muscular efforts, the slow fibers are recruited first.
- They contain myoglobin which is similar to hemoglobin that it combines with oxygen and store it in the muscle cell until it is needed for mitochondria.
- Therefore, the slow fibers are ideal for low intensity, endurance activity such as quiet breathing, have high metabolic capacity, and specialized in yielding aerobic energy.

- However, if subjected to sub-maximal activity, they also exhaust their glycogen stores and show fatigue.

Type II: Fast Twitch Fibers

- These fibers are more adapted for strength.
- They have more extensive sarcoplasmic reticulum, thus calcium can be rapidly released and taken up allowing rapid contraction.
- They reach peak tension in a very short time, when high intensity respiratory activity such as vigorous coughing is needed.
- However, they have low oxidative capacity and are very susceptible for fatigue when repeatedly activated.

Means to Increase the Strength and Endurance

It has been established that it is necessary to increase the strength and endurance of the respiratory muscles in the process of weaning.

Strength

- **T-piece trials** to the patient being weaned, is one *method of increasing* ***the strength***.
- In this, *a short burst of repetitive intense activity* is allowed.
- This type of activity causes the muscle size to increase (hypertrophy).
- The individual muscle fiber increase in diameter, their nutrient store increases, they even gain in mitochondria.
- Both their motive power and their nutrient mechanisms for supporting increases.
- However, patients with low compliance and increased resistance due to the ventilatory circuit may find the energy requirement for this work as too much and experience fatigue.

- Under these circumstances, it is mandatory to restrict the duration of the trial and resort to strategies designed to decrease the work of breathing.

Endurance

- For enhancing ***the endurance***, numerous *repetitions of low intensity activity* is advised.
- Using pressure support mode for the patient being weaned is a good method of increasing endurance.
- This less intense muscular activity sustained for long periods will promote muscle endurance, not hypertrophy.
- It promotes mitochondrial enhancement and increases oxidative enzymes, myoglobin and even the number of blood capillaries to support increased oxygen uptake and muscle metabolism.

IDENTIFYING MUSCLE FATIGUE, PREVENTION, AND MANAGEMENT

Fatigue is defined as the inability of the muscle to continue to develop the force sufficient to perform a particular work.

- In the process of weaning, muscle fatigue may be caused by excessive exertion by unduly pronged periods of assuming full work of breathing by the patient must be avoided.
- Therefore, the respiratory muscle fatigue is the inability of the muscles to produce sufficient pressure changes to maintain adequate alveolar ventilation which can be life threatening.

Causes and Prevention

- Maintaining of a balance between **demand** and **supply** factors related to the muscles is essential.

- *Demand factors* include the work of breathing and the intensity of muscle contraction required for the expected work.
- *Supply factors* include the provision and utilisation of necessary substrates, such as the blood flow to the muscle, oxygen content in the blood, general nutritional status, glycogen stores, and the muscles ability to utilise the energy (metabolic status).
- Increase in the energy demand may be seen in increased resistance and reduced compliance.
- Anemia, hypoxemia, low cardiac output conditions, hypophosphataemia, electrolyte imbalance, (particularly sodium, potassium, and chloride) and starvation are some of the conditions which cause imbalance.
- Particularly, *when the blood supply to the diaphragm is reduced* in shock or low cardiac output states, the force of contraction diminishes.
- Increased energy demand in the muscles will deplete muscle energy stores of glycogen, adenosine triphosphate (ATP) will certainly result in muscle fatigue.
- An accumulation of metabolic products particularly lactic acid which commonly results in ischemic muscles with low perfusion and a decrease in pH cause muscle fatigue.

Clinical Signs of Muscle Fatigue

- Increase in respiratory rate.
- Rapid shallow breathing with a minute volume (V_E) in excess of 10 L/ min.
- Though the V_E is more, the alveolar ventilation is reduced because the inspiratory flow is mainly happening in the dead space.

- As the patient continues to breathe under fatiguing conditions, bradypnea and central apnea will ensue. The exact mechanism is ill understood.
- Observation of abnormal breathing mechanics (abdominal paradox) will precede an increase in $PaCO_2$, thus increase in $PaCO_2$ is a late sign of fatigue.

So, when fatigue begins, monitoring of respiratory rate (RR), minute volume (V_E), tidal volume (V_T) and breathing pattern will signal the changes that precede changes in pH and $PaCO_2$.

- When the patient has weakness of muscles, exercise training to develop strength and endurance is needed.
- However, when the muscle fatigue is the cause of weakness, then rest for muscle is required and not training.
- It is to be remembered that it is *reversible by ensuring adequate energy supply to the muscle and by rest.*

Rest to Muscles

In fact, in the process of weaning, the physician needs to have greater degree of patience than that is required for the patient, particularly weaning a patient after prolonged artificial ventilation.

- For successful weaning, gradual reconvicting of the respiratory muscles, periods of exercise should be alternated with complete rest.
- Rest and nutrition are essential to replenish the necessary muscle glycogen and ATP.
- Therefore, *adequate rest and prevention of fatigue are the corner stones in the weaning program (respiratory muscle training program).*
- Now returning the patient to a mode that assumes the work of breathing (WOB) will allow the respiratory muscles to rest.

- It is interesting to recall that the metabolic cost (oxygen requirement) of normal spontaneous breathing is only 1% to 3% of total body oxygen consumption.
- Whereas oxygen necessary to support ventilation in weaning trials may rise as high as 25% of the total oxygen consumption.
- The best mode for this purpose is not well defined, but assist/control (A/C) and high level pressure support (PS) may be suitable.
- The amount of time given for rest can be gradually decreased as the patient's muscle power improves.

WEANING CRITERIA

The readiness of the patient to be weaned has to be assessed, before weaning process is started. This can be done based on certain clinical criteria and values of parameters. Various authorities have proposed different criteria for this purpose. However, the idea of looking into the criteria is to *avoid premature weaning trials.*

Therefore, weaning criteria is theoretically providing information as to whether the patient can be successfully weaned. The predictions made on these criteria may not be absolutely correct, because of the practical difficulties and the need for the patient's cooperation in many of the tests. However, it gives an insight of the dependence of the patient.

If weaning is attempted before it is apt to do, then it may impose severe stress on the cardiorespiratory system leading on to prolonged support with its own complications.

Weaning criteria is likely to provide information about three aspects of respiration. They are *ventilation, oxygenation, and respiratory mechanics*.

Ventilation

Adequacy of ventilation can be assessed by values of blood gas analysis.

- $PaCO_2$ value must be normal: **35 mm to 45 mm Hg**.
- pH value must be: **7.3 to 7.45**
- V_D/V_T ratio must be: **< 0.6**

  V_D/V_T ratio: of more than **0. 6 indicates** that the ventilator reserve is less than the demand.

Oxygenation

Oxygenation can be assessed by:

- PaO_2 value of **> 70 mm Hg** on a F_IO_2 of **< 0.5** with PEEP of **5 cm H_2O** or less.
- PaO_2/F_IO_2 ratio should be more than 200.
- A value of the ratio less than 200 indicates that the intrapulmonary shunt (Q_S/Q_T) is about 20% and the patient not ready to be weaned.

Mechanics

The evaluation of mechanics tries to quantify the muscle function in terms of **strength** and endurance**.** The parameters assessed are:

- Tidal volume (V_T)
- Minute ventilation (V_E)
- Maximum voluntary ventilation (MVV)
- Vital capacity (VC)
- Maximum inspiratory pressure (MIP).

Minute Ventilation (V_E)

- This can be measured by connecting the hand held spirometer (Wrights Respirometer) to the endotracheal

tube and asking the patient to breathe normally for one minute.

- The normal value is about 6 L/min.
- Values between > 5 L/min but < 10 L/Min are desirable for considering weaning trials.
- Requirement of a high V_E to maintain acceptable $PaCO_2$, places an increased workload on the respiratory muscles.
- V_E value > 10 L/min, together with rapid shallow breathing is associated with respiratory muscle fatigue.

Tidal Volume

- Minute volume (V_E) is measured with a Wrights Respirometer and that is divided by the respiratory rate will give the value of tidal volume (V_T) and is mentioned in ml.
- The average value is > 300 ml less than 300 ml indicates that patient will have difficulty in maintaining adequate alveolar ventilation.
- More meaningful way expressing it is **> 5 ml/kg** (normal value is **5 to 8 ml/kg**).
- Tidal volume is considered as an index of respiratory muscle endurance.

Maximum voluntary ventilation (MVV): (Maximum breathing capacity)

- This can be calculated by the Wrights Respirometer attached to the endotracheal tube and instructing the patient to breathe as hard and fast as possible for one minute.
- This gives an idea about the patient's ability to sustain ventilation under stress.
- Normal value is **50 to 250 L/min.**

- The relation between the V_E and MVV gives a measure of ventilatory reserve and muscle endurance.
- If the patient about to be weaned has a value which is more than double the V_E can go for weaning trial.
- This procedure is really tiring to the patient and may exhaust him and it needs extreme cooperation from the patient.
- It may give unreliable values while the normal range itself is so wide.

Vital Capacity

- Vital capacity is maximum volume exhaled after maximum inspiration.
- It is a measurement of ventilatory reserve and strength.
- It reflects the patient's ability to take deep breath, cough, clear secretions, and reverse atelectasis. Cough is one of the best methods of recruiting collapsed alveoli with normal time constant.
- Normal value is **65 to 75 ml/kg.**
- A value of **< 10 to 15 ml/kg** indicates that the patient will become fatigued. He will not be able to cough, sigh and so atelectatic alveoli will not open up.
- It can be measured by a Wrights Respirometer, but educating the patient is needed and his cooperation for performing the test. It is not very reliable.

Maximum Inspiratory Pressure

- This is sometimes is known by other terms, negative inspiratory pressure (NIP), negative inspiratory force (NIF), maximum inspiratory force (MIF), peak negative pressure (PNP), inspiratory force (IF) and so on.
- This measurement indicates the overall strength of respiratory muscles and ventilatory reserve.

- This also indicates the ability to take deep breath and generate adequate intrathoracic pressure for effective cough.
- In normal healthy individuals it is about **– 115 cm H_2O**. (About 25% lower values in women.)
- A value of > 30 cm H_2O is considered safe for weaning.
- It can be measured by the hand held equipment known as "Inspiratory force meter" which can be connected to the endotracheal tube. The patient is instructed to do normal exhalation (to FRC) and then to inspire with maximal attainable force against an occluded airway. The occlusion is maintained for 5 to 10 seconds unless this degree of negativity is contraindicated.

Respiratory Rate

- The respiratory rate is one of most sensitive index of readiness for weaning. Unfortunately due importance is not given to this while considering weaning.
- *It is a single factor which correlates with the ability to sustain spontaneous breathing.*
- It must be counted for one full minute. Even if the digital read out is present in the ventilator when the patient is in CPAP, it should not be relied on.
- Because, the respiratory pattern may change within one minute, it has to be counted by looking at the chest wall movements.
- Normal rate is 12 to 20/min.
- Any rate > 25 indicates that patient has inadequate muscle strength to achieve adequate tidal volume.
- Other factors that may increase the respiratory rate such as fear, anxiety, pain, fever, hypoxemia, hypercarbia must be kept in mind.

- *Any rate > 35 cannot be sustained and it indicates respiratory muscle fatigue.*

WEANING MODES

It is not very easy to choose the right mode for weaning program, as it varies with each individual patient. So here, it may be discussed, how the different modes are used for weaning and their merits and demerits. Though a few modes are recommended for weaning, no one is found to be superior to others.

Some of the modes used are; T- piece trials, CPAP, SIMV, PSV and MMV.

- The weaning program must aim at *meeting the ventilatory demand* of the patient to *achieve optimum ventilatory function* at the same time, *preventing fatigue* of the respiratory muscles.
- The main considerations are comfort, synchrony, and work load application. In spite of careful application, one is very uncertain about the outcome.
- The amount of work transferred to the patient must be at a tolerable level and the balance of the work is done by ventilator.

T-piece Trial

- This is the original method of weaning till IMV was introduced in early 1970s.
- In this method, the patient is disconnected from the ventilator to breathe spontaneously humidified oxygen for varying periods.
- Between these T-piece trials, the patient is re-connected to ventilator and given full support.
- This is actually fully loaded spontaneous breathings are alternated with fully supported mechanical ventilation.

- This type of work improves the respiratory *muscle strength.*
- As the trials increase in duration, it will enhance the *respiratory muscle endurance*.
- T-piece trials are indicated for patients with a reliable respiratory drive.
- The *advantage of T-piece trial is that the work of breathing may actually be reduced* when compared to CPAP or SIMV because there are *no demand valves to open or ventilatory circuit to breathe through.*
- It can be started with the T-piece set up with the same F_IO_2 as in the ventilator or 10% more than that set in the ventilator.
- First period of spontaneous ventilation may be as short as 5 minutes.
- Periods of spontaneous breathing become progressively longer when the patient is able to take more work of breathing. As the duration increases the frequency decreases.
- The frequency of weaning may vary; it can be two to six per day. However, it totally depends upon the individual patient, how much of rest he requires between trials.
- Once the patient is able to breathe spontaneously for the whole of day time, then night trial may be started.
- When the patient is on “No support” for more than 24 hours, support can be withdrawn and extubation planned.

CPAP Trial

- This trial is done when the patient is still on ventilator.
- This trial is suited for patients who are at risk of hypoxemia due to atelectasis.

- CPAP improves alveolar stability, increases the FRC, and improves distribution of ventilation in smaller airways.
- It is indicated in patients in whom oxygen saturation decreases during spontaneous breathing.
- In patients, where other weaning parameters are satisfactory, but PEEP is still required to maintain oxygenation, it has definite place.
- The WOB actually increases, as the patient has to exhale against the positive airway pressure.
- After the trial period patient is returned to full support such as PS or A/C mode.
- Initial trial of spontaneous breathing with CPAP may be for a period of 5 minutes.
- Periods of spontaneous breathing become progressively longer, as the patient's respiratory mechanics improves and is able to take more of work of breathing.
- The frequency of weaning trials again depends upon the patient's condition as to how much of rest he needs in between the trials. It may be from two to six per day.
- Once the patient is on spontaneous ventilation with CPAP for the entire day time, then nighttime weaning can be started.
- This alternating CPAP and full supported ventilation is continued till patient is able to do sustained spontaneous ventilation.

SIMV Trial

- Initially Intermittent mandatory ventilation (IMV) was introduced as a weaning mode in 1970s.
- This wax later modified as SIMV as the mandatory breaths were perfectly synchronised with the inspiratory efforts of the patient thus making the patient comfortable.

- When compared to T-piece and CPAP trials, this is more suited for patients who are anxious about their ability to breathe with out ventilator.
- This mode allows the patient to breath spontaneously in between the mechanical breaths.
- Weaning is done by gradually reducing the mandatory breaths by 1 to 2 breaths per minute.
- The patient's tolerance for the new level of support is assessed before any further new reduction is done.
- Once the reduction has reached 2 to 4 breaths per minute, a trial with spontaneous breathing on CPAP or T-piece may be tried followed by extubation.
- In this weaning, the patient is alternated with fully loads spontaneous breaths by fully supported mechanical breaths over a few breathing cycles.

Pressure Support (PS) Trial

- Pressure support was introduced in 1980s as a ventilatory support and weaning mode.
- Every breath is augmented by pressure assistance; so the patient does not need to take any fully loaded spontaneous breaths.
- By providing inspiratory flow assistance, internal impedance loads are overcome.
- Pressure work is decreased as the tidal volumes are augmented.
- Hence, the work performed by the patient is a high volume, low pressure quality that will promote the endurance conditioning of the respiratory muscles.

Advantages

- When the PS level is gradually reduced, the work load taken up by the patient gradually increases.

- The oxygen consumption is reduced than the other weaning modes.
- It is advantageous in patient with reduced cardiac reserve and the patients who do not have enough muscle strength to take over the WOB which is required in other modes like T-piece trials, CPAP trials, and SIMV trials.
- This improves the patient comfort and tolerance because the patient has control over thee respiratory rate, inspiratory flow, tidal volumes, and inspiratory time. Patient has greater control over his respiratory cycle.
- Low level PS such as 5 to 7 cm H_2O is useful in overcoming inspiratory resistance caused by the endotracheal tube and the ventilator circuit.
- PS can be used alone or with SIMV.
- ***PS alone:*** Begin with a level of PS that ensures a tidal volume (V_T) of 5 ml/kg and a respiratory rate (RR) of 25/min.
- Reduce the PS by 2 to 5 cm increments at the same time me assessing the patient's tolerance of reduced level of ventilatory assistance.
- When the PS level is reduced, if the lung mechanics are stable, the respiratory rate (RR) should not increase substantially and the tidal volume (V_T) must remain around 5 ml/kg.
- Weaning progresses till the patient is brought down to PS level of 5 cm H_2O, which is enough to overcome the inspiratory resistance of the circuit.
- Once the patient is able to breath with low level PS all the day time, then night time weaning may be started.
- Complete rest between the trials is given with either PS_{MAX} – (a level that gives full ventilatory support V_T of 10 to 12 ml/kg) or A/C mode.

- ***PS and SIMV:*** The PS is used to assist the spontaneous respiration of inadequate tidal volume (V_T) or to overcome the resistance of demand valve or endotracheal tube.
- Weaning is accomplished by decreasing the number of SIMV breaths and then a gradual reduction in PS level.

Psychological Aspect of Weaning

- Preparing the patient psychologically to come out of ventilator is the primary essential step of weaning.
- The patient must be approached with compassion, empathy, and patience.
- Stay with the patient initially and communicate in a calm and reassuring manner. Explain the weaning procedure as often as necessary and reduce anxiety.
- When a patient is alert and well oriented, certainly he suffers from anxiety and fear of withdrawal of support that he was receiving so far, which maintained his life.
- He must be strongly reassured that if he feels any difficulty and is unable to continue, he will be supported adequately before fatigue develops.
- He must be informed about the weaning plan and he has to be explained that weaning is an exercise training program for the respiratory muscles that they will be gradually taking over more of the WOB.
- He must be informed that he has to give feed back about how he feels physically and emotionally throughout the weaning.
- The physician must develop a mode of communication with the patient that he effectively participates mostly by signs and facial expressions.

Facts to be Remembered

- Assess the patient and make sure that he is ready for weaning.

- Always weaning is started in the beginning of the day.
- It must be restricted to waking hours.
- Weaning is given the first priority and should not be done after any energy depleting activity like bathing, chest physiotherapy, suctioning, etc.
- Preferably patient must be in a semi-Fowler's position that allows full excursions of diaphragm without limitation by abdominal organs.
- Suctioning of the airway done and allow enough time for the patients vital signs to return to the baseline after the suctioning procedure.
- Perform the baseline assessment of cardiac, pulmonary, and neurologic function.
- To increase the confidence, provide positive, supportive fedback about how well he is doing.
- Perform ongoing assessment of patient's tolerance. Assess for signs and symptoms that the WOB is too much and the patient is beginning to fatigue.

Signs for Reinstituting Mechanical Ventilation

Respiratory

- Respiratory rate (RR) exceeding 30 to 35 per minute.
- Abnormal respiratory pattern.
- Asynchrony of thoracic cage and abdomen.
- Uses of accessory muscles.
- Exhaled (V_T) tidal volume is < 5 ml/kg.
- Decreased SaO_2.
- Increasing $EtCO_2$.

Cardiovascular

- Heart rate change of 20 beats/minute.
- Change in blood pressure of 20 mm Hg or more.

- Ventricular ectopic more than 6 per minute.
- Skin temperature increasing and sweating.

Neurological Changes

- Anxiety
- Confusion
- Agitation
- Somnolence indicating hypoxemia or hypercapnea.
- Good clinical assessment should be utilised to determine to say when the patient is beginning to fatigue and connect him back to ventilator.
- It is important to see that the patient is not allowed to get exhausted that the muscle energy stores are depleted.

There are very many weaning criteria suggested by various authorities. Essentially all aim at one goal. Some are discussed for the reader to analyse.

SIMPLE WEANING CRITERIA FOR ICU PATIENTS (A-E)

A. **Alert:** Alert, cooperative, and has a stable medical status.

B. **Breathing rate and depth:** Spontaneous respiratory rate (RR) (f), and tidal volume (V_T).
f/V_T ratio must be **< 105**

C. **Cough:** Vigorous cough, sufficient enough to clear the secretions and maintain a patent airway.

D. **Durability** (Endurance)
   - Adequate and sustained muscle strength.
   - This may be assessed by any number of parameters such as negative inspiratory force, maximum minute ventilation, sustained head lift, and documented absence of residual NMB.
   - In addition some authors suggest 2 hour trial on T piece breathing system.

"O_2 must be as high as necessary and as low as possible".

E. Efficient oxygenation.
$PaCO_2/F_IO_2$ ratio must be **> 200.**
PEEP < 5 cm H_2O.

WEANING STATUS OF PARAMETERS

• Vital Capacity (VC):	**>10–15 ml/kg**
• Negative inspiratory effort:	**> 30 cm H_2O**
• Minute ventilation:	**< 10 Lit./min**
• PaO2:	**> 60 mm Hg with FiO_2 < 0.4**
• PaCO2:	**< 45 mm Hg**

A. Parameters commonly used to predict successful weaning:

1. Adequacy of oxygenation:	PaO_2/F_IO_2 > 200–250 **or** PaO_2 > 100 mm Hg on < 5 cm PEEP and F_IO_2 < 0.4, Pulmonary shunt (QS/Q_T), 20%.
2. Ventilatory mechanics:	Vital Capacity (VC) at least 10–15 ml/kg. Tidal Volume (V_T) > 5 ml/kg; Static systemic compliance > 30 ml/cm H_2O.
3. Respiratory muscle strength:	Negative Inspiratory Force – 20–30 cm H_2O.
4. Ventilatory demand:	< 10 Lit/minute volume (V_E) required for maintaining PCO_2 of 40 mm Hg. Dead space to Tidal volume ratio – (V_D/V_T) < 0.6.

5. Ventilatory reserve: Maximal voluntary Ventilation > twice the Minute volume (V_E) required for a $PaCO_2$ of 40 mm Hg.

* Airway pathology, * Blood volume, * Hemoglobin, * Hemodynamics, * Level of consciousness, *Pulmonary secretions, * Temperature, *Surgical pain, * Emotional status, * Nutrition, * Electrolytes. All these can modify the condition.

B. If weaning parameters are inadequate:

Careful search for reversible pulmonary or systemic pathology must be made.

- *Inadequate oxygenation* or a large QS /Q_T may result from:
  Lung atelectasis: *Collapse, *Pneumonia, * Aspiration, *Acute lung injury.
- *Inadequate ventilation:* High PCO_2 may result from: Drug induced respiratory depression, * Excessive VCO_2 (Fever, shivering, high respiratory quotient) *High V_D/ V_T (ARDS) or Pulmonary embolism, * Bronchospasm.
- *Respiratory muscle weakness:*
  May result from:* Residual NMB, * Electrolyte Disorders; low phosphate, low or high magnesium, low potassium, * Malnutrition, * Neuromuscular disease, * Reduced blood flow to the diaphragm due to hypovolemia, * Cardiogenic or Septic shock.
- *Ventilatory failure:*
  Can occur due to* Increased work of breathing due to increased airway resistance, e.g. Bronchospasm, * Small endotracheal tube, * Low compliance.

C. The therapy depends on the underlying pathology:

- IPPV can reduce:
  - The work of breathing.
  - Improve distribution of ventilation.

  - Improve the gas exchange.
- PEEP or CPAP can improve oxygenation by increasing the FRC and V/Q matching.

D. When the weaning parameters predict successful weaning:

A few means of ventilator discontinuance used may be:
- T Piece
- IMV
- PSV
- MMV.

During weaning, the indicators that suggest mechanical ventilation is needed are:
- Deterioration of ABG
- Rapid rate > 30/min
- Paradoxical breathing
- Hemodynamic changes.

E. Inability to wean despite optimization of the patient's physiology may indicate the need for tracheostomy and chronic ventilatory support.

F. Once ready for extubation:
- The patient is placed on 100% O_2.
- Trachea and pharynx are suctioned.
- Endotracheal tube cuff is deflated.
- Endotracheal tube is pulled out gently during expiration *(During expiration vocal cords are wide apart and less damage is caused to them by the tube).*
- Close monitoring continued for several hours.

G. Reintubation is done when:
- Upper airway obstruction is noted.
- For control of tracheo-bronchial secretions.
- Reappearance of respiratory failure.

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